New Orleans Medical
and
Surgical Journal

Vol. 100 JUNE, 1948 No. 12

CLINICAL OBSERVATIONS ON THE TREATMENT OF THE ALCOHOLIC*

ROY CARL YOUNG, M. D.†
COVINGTON

This report is based on the study and treatment of 2275 sanitarium cases of alcoholism, the majority of which were observed and studied for at least four weeks. It expresses some of the observations that I have made in the management, study and treatment of the alcoholic patient.

The alcoholic suits admirably the caption, "Sick in body and sick in mind." His case is primarily one for the psychiatrist, as alcoholism has its basic starting point in the psychic sphere. Contributing causes may be timidity, bashfulness, feelings of inadequacy or inferiority, or deeper personality maladjustments.

It is at this initial stage that psychiatrists prefer to come in contact with the case. Psychiatric consultations and guidance for these patients would prevent the progress of emotional problems, and, with proper insight and confidence restored, there would not be the resultant tensions and somatic symptoms that so often lead to a flight into alcoholism as a temporary or permanent escape mechanism.

The younger an addict started drinking the harder will be the problem of cure, possibly because of alcohol's effect on immature emotions. I have in mind those patients who started drinking during their early high school days. A history of inebriety at the age of fourteen, fifteen and sixteen is not an infrequent occurrence.

The development of the alcoholic is, as a rule, not rapid until a certain point is reached. Alcoholism then develops with alarming rapidity. In my observation, this point is reached when the narcotic effect of the alcohol is sought. The victim then drinks to blot out, temporarily at least, all conscious memories. Frequently, under the effects of the drug, as a result of continued drinking, the waking and passing out either ends disastrously or toxic symptoms develop, such as hallucinosis, delirium tremens, partial starvation, and acute avitaminosis.

Psychiatry is the door to the hidden forces that cause the patient to become an alcoholic addict. It is my conclusion that each alcoholic patient can give the psychiatrist all the letters of the alphabet necessary to spell success in his case. There may be exceptions, but in my clinical experience these are rare. I do not mean to imply that these cases are as simple as A.B.C., but I do believe that, during the course of several weeks to two months, the psychiatrist will find the key to the case, provided the alcoholic patient is willing to cooperate and really wants the help the psychiatrist can give him. Many times I have seen a patient who would not frankly discuss his problems—even deny things I knew positively were true—and later take the attitude that he had fooled the physician. This non-cooperation on the part of the patient, even though cooperating in all other re-

*Read before Southern Psychiatric Association, October 13-14, 1947, Birmingham, Alabama.

†Medical Director Fenwick Sanitarium, Covington, Louisiana.

spects, cannot result in anything except failure of the patient to make an adjustment. Every psychiatrist can help every alcoholic if that alcoholic really wants help.

We, as psychiatrists, can understand these mental mechanisms of non-cooperation, but at times it is extremely difficult to get the patient to give his full response. It requires tact, diplomacy, and training, and sometimes one wonders, "Who the devil wants to be a diplomat!" The non-cooperative type will often respond to psychiatric studies after two or three weeks, whereas psychiatrically speaking prior to that time they were "untouchables." These are usually cases of constitutional inferiorities with emotional immaturity, psychoneurosis with inferiority complexes and over-compensations, and, last but not least, the schizoid personalities.

At this point we should differentiate between emotional immaturity and emotional instability resulting from the alcoholic spree or debauch. The former has never grown up emotionally and the latter is emotionally unstable as a result of an overdose of alcohol and side reactions resulting therefrom. The emotionally immature patient will often respond and cooperate if the daily visit is made for some time on a neutral ground. This type is suspicious and often thinks the psychiatrist is trying to prove that something is mentally wrong. The patient, however, will often become friendly with the physician in a few days, and with tact the psychiatric aspects of the case can be obtained; the same patient, however, had he known the physician was a psychiatrist, would not have opened up. I have had this experience several times and have been told by some of the cases that I should study psychiatry. The point is that many alcoholics are "gun shy" of psychiatrists per se but will often cooperate fully with the physician-psychiatrist.

On the other hand, there is the alcoholic who demands a psychiatrist at once and expects the psychiatrist to convince him that he is not an alcoholic and point out a clear course by which he can return to drinking socially without becoming intoxicated. Such a course does not exist, because the alcoholic cannot remain well except by total abstinence forever, and he must never revert to social drinking.

I have seen such patients come in and profess most sincerely and earnestly that they want to get well at any cost or sacrifice. This is especially true when they are accompanied by relatives or close friends. Statements of this kind made when the patient is on the verge of cracking up, or just getting over a binge during which he causes a great deal of worry and anxiety, should not be taken too seriously, as frequently this is an effort to have the parents and friends feel sorry for him as a result of his predicament. The statements and promises made at the end of several weeks when he is returning to a normal basis are more dependable. The shame, remorse and regret manifested on admission often disappear in two or three days as the patient feels better; then we see the individual picturing his episode in a comical way, with himself as the chief comedian or hero, as the case may be. Very seldom is the sordid side ever told; incidents may be admitted, however, if the information is from some other source than the patient, but never elaborated on.

The alcoholic or the ex-alcoholic is always hoping that the time will come when he can resume drinking alcoholic beverages socially. This is a fervent hope and wish of these individuals and *this is the most frequent cause of relapse.* The false belief that some day they will again try to prove that they can control their drinking is in the mind of the majority of alcoholics. Sooner or later they will put the theory to a test and—bingo! It is certainly bad personal psychology for the patient to carry this thought in mind. It acts as a constant suggestion. On the other hand if he accepts the law that, under all circumstances, he can never drink, then the mental hazard is eliminated.

Beware the alcoholic patient who gets well in five or six days, goes around telling everyone how wonderful he feels, what a great treatment he received, that the world

is rosy in every respect, and there will be no more drinks for him. He is riding on a wave of euphoric purity based on emotionalism and not on stability of the central nervous system. He'll be drunk again in six weeks or sooner.

Many alcoholics are proud of the fact that they are alcoholics and brag about the many treatments they have taken and the many psychiatrists they have seen and they end with the lament that no one ever cured them. They often use the fact that they were treated before as proof that they are incurable. Some alcoholics want to be considered incurable and above all want their family to consider them so, in order that they may continue their indulgence without interruption or interference. It matters not that they attempt to drive while intoxicated and endanger the lives of others.

All sanitariums have a "repeater" patient who is always telling the new patient about the various times he has been admitted and the several institutions that he has been to; he knows all the methods used and all the answers to the alcoholic problem, and he usually throws the "wet blanket" on the new patient by saying, "You'll go back to drinking and be back in a year; they all do." This is purely an alibi for his own failures, but this line of personal propaganda has to be combatted and defeated.

At this time let me say a word of praise for that fine group of alcoholics who are sincere in wanting to get well and who are most cooperative. They get well and stay well, and they are among our best citizens. They need no defense from any one. They have been sick and have recovered.

The alcoholic patient often has many physical defects, both physical and neurological, and, until these are corrected, the patient cannot be considered well or in a position to resume his occupation. The periodic alcoholic who goes on a binge every three or four weeks usually consumes large quantities of alcohol for a period of several days and stops only when he is so sick that nothing will remain in his stomach. He is then willing for the physician to help him. These periods of near starvation are bound to produce a nutritional and vitamin imbalance of the worst type, as well as dysfunctions or disturbed functions of the stomach, such as gastritis, hepatitis, and malnutrition of brain cells, with the resultant emotional instability. Consider what will eventually happen over a period of a year or more if these periods of alcoholic forced starvation are repeated every few weeks. The individual becomes more and more addicted to the narcotic effect of the alcohol and, therefore, he more often repeats the alcoholic spree because the addiction becomes stronger. The nutritional state grows worse, the vitamin deficiency more and more pronounced, with a resulting chain of symptoms referred to the varying systems. Not the least is the change of personality that frequently results.

This is true of the steady or daily drinker who, over a period of years, consumes from a pint to a quart of whiskey daily. Changes occur in his system, a result of the nutritional imbalance resulting from the alcoholism. I am not speaking of the chronic alcoholic who is drunk daily from this amount, but I have reference to the man who drinks from a pint to a quart and who is daily at his work. It is very difficult to make these patients understand that time and proper treatment are necessary to repair or correct the physical damage that they have incurred as a result of the vitamin and nutritional changes caused by their drinking. Many research physicians and clinicians assert that the damage is a result of the inhibitory action of the alcohol on the absorption of vitamins, or that the changes occur from the impaired nutritional condition resulting from the ingestion of alcohol.

Goodhart and Jolliffe,[1] in their discussion of polyneuritis of alcohol and the dose of Vitamin B_1, quote an article by Jolliffe, Colbert and Joffe[2] "in which they found that the diets of the alcoholic addict with polyneuritis failed over an effective period of time to contain an adequate quantity of

Vitamin B; and second, that the diets of alcoholic addicts without polyneuritis, though the addiction was of long duration, contained adequate quantity of Vitamin B; and third, that certain subjects without polyneuritis consumed enough alcohol over a sufficient period of time to cause peripheral nerve impairment if alcohol per se was its cause."

Jolliffe and Goodhart speak of the lack of improvement in the objective signs of motor nerve involvement after treatment and they believe it to be caused by relatively "Irreversible anatomic changes in the peripheral nerves occasioned by an acute Vitamin B_1 deficiency." They conclude: "Vitamin B deficiency is the primary cause of the polyneuritis of the alcoholic addict."

Piker and Cohn,[3] reporting on the treatment of delirium tremens cited much improvement in statistics at Cincinnati General Hospital with a treatment of spinal drainage, 50 c.c. 50 per cent glucose intravenously four to five times daily, digitalization within twenty-four to thirty-six hours, sedation using paraldehyde, and a high caloric intake supplemented by vitamins of the B group. The mortality rate was dropped to 5.3 per cent and the average hospital stay was 4.8 days. This report is based on a study of 300 consecutive cases admitted to the Cincinnati General Hospital from January 1, 1933, to July 1, 1935.

Spies, Chinn and McLester,[4] in their studies on "Severe Endemic Pellagra", conclude, "The signs and symptoms, the predisposing dietary insufficiency, the clinical course of the disease and the response to treatment of these patients with endemic pellagra was the same as those of pellagrins previously studied whose disease followed the chronic excessive use of alcohol, the so-called alcoholic pellagra."

Spies, Cooper, and Blankenhorn[5] reporting on the use of nicotinic acid in the treatment of pellagra conclude: "The lesions of the mucous membrane in 11 cases of pellagra (2 endemic pellagra, 3 alcoholic pellagra and 6 pellagras secondary to organic disease) were cured promptly by the administration of nicotinic acid."

Askey[6] writes: "Vitamin deficiencies in chronic alcoholism may be brought about by an inadequate diet, insufficient vitamins, deranged gastro-intestinal function (affecting both digestion and absorption) and a diminution in the volume of the gastric secretion, a diminished acidity and an increased incidence of achlorhydria." He asserts, "It is impossible to predict when clinical manifestations of pellagra or those of polyneuritis will develop in a patient with chronic alcoholism." He stresses the multiple vitamin deficiences rather than one particular vitamin and further emphasises the necessity of a high protein diet as well as vitamins.

Jolliffe, Bowman, Rosenblum and Fein[7] report 150 cases of an encephalopathic syndrome, a condition heretofore nearly always fatal, which they believe was caused by nicotinic acid deficiency. The above authors quote Bender and Schilder who describe the clinical picture as one of a group of five which they called "encephalopathia alcoholica". Their classification of the type of alcoholics encephalopathia was related to the most pronounced manifestations: No. 1, Clouding of consciousness and changing rigidities. No. 2, Cerebellar symptoms. No. 3, Catatonia. No. 4, Alcoholic delirium. No. 5, Polyneuritis. Further in the article they state "The encephalopathic syndrome does not occur exclusively in alcoholic patients" and they quote Spies and Sydenstrecker "as having observed this syndrome in endemic pellagrins."

Foreign Letters A. M. A. reporting on the Third International Congress on Nutrition:[8] "In the discussions attention was urgently directed to the extremely harmful effects of chronic alcoholism biologically, morally, and socially on the under-nourished populations."

Conclusions by Spies and Vilter on Vitamin E:[9] "Our findings suggest that an injection of synthetic alphotocopherol is effective temporarily in relieving neuromuscular symptoms, roaring sensations in the ears, anorexia and insomnia in selected persons with malnutrition, but with no

clinical evidence of pellagra, beriberi or riboflavin deficiencies."

Kiene, Streitweiser and Miller[10] in their paper state that their primary purpose was to prove that alcohol is not the principal factor in the production of delirium tremens and that a deficit of Vitamin B_1 content in the brain parenchyma is chiefly responsible. A central neuronitis, an irritative phenomena, is caused by a perverted carbohydrate and Vitamin B_1 relationship, which when treated with adequate Vitamin B_1 responds more remarkably, as far as subsidence of symptoms is concerned, than do the peripheral states of polyneuritis of alcoholic origin.

Robinson and Shelton,[11] reporting on the use of insulin, glucose and thiamine chloride in the treatment of acute alcoholism, base their treatment on the belief that alcoholism is a disease with a chronic progressive pathological background, characterized by successive acute pathophysiologic episodes which have primarily a nutritional background.

An editorial in the A. M. A.[12] states that Jolliffe has shown that the rather narrow margin of safety in the modern American diet with respect to the adequacy of Vitamin B_1 can be greatly decreased or largely erased by the consumption of alcohol, since the metabolism of the alcohol calories increases the need for thiamine which the alcohol itself cannot provide.

Jolliffe[13] concludes from his studies that (a) no alcohol addict with an estimated adequate vitamin intake had polyneuritis and (b) every alcohol addict with an estimated absolute deficiency of Vitamin B for twenty-one days or more had polyneuritis. Polyneuritis may develop as early as the seventh day in an alcohol addict of estimated vitamin deficiency.

Queries and Minor Notes A. M. A.[14] has this to say: "Vitamins in chronic alcoholism. Strictly speaking vitamin therapy is not a treatment of alcoholism, but is frequently of value in the treatment of the hypo-avitaminosis that accompanies chronic alcoholic addiction. This lack of vitamins is chiefly the result of the failure of the addicts to eat sufficient vitamin containing food. While the failure results in a lack of the food contained vitamins, the most common clinical indication of vitamin lack is the occurrence of peripheral neuritis."

Alexander[15] describing the changes in the nervous system of chronic alcoholics states "The majority of pathological conditions encountered in the nervous system in chronic alcoholism are attributable to associated vitamin deficiencies, particularly Vitamin B_1, nicotinic acid and Vitamin C."

Bean, Spies and Blankenhorn[16] in writing on pellaga in Ohio Hospitals state, "In alcoholic pellagra the vitamin wants have not been satisfied because the beverage alcohol, the chief source of calories, ordinarily contains none and the diet does not make up the deficit."

Strecker and Rivers[17] report the results in 133 cases of alcoholic toxic states with 100 c.c. 50 per cent dextrose, 30 units of insulin and 100 mgs. of thiamine hydrochloride. This can be repeated in three hours if necessary. Results were excellent.

Cannon, Modarelli, DeVincenzo and Swiney[18] report, "The treatment of 15 cases of delirium tremens with an intravenous injection of 50 c.c. of 50 per cent glucose, 8 c.c. of Vitamin B_1, approximately 25000 units, and 25 units of insulin as soon as the diagnosis is made. In the majority of cases only one injection is necessary. The improvement is due to a decrease in cerebral edema as well as to supplying the vitamin deficiency and the increased nutrition for the brain contained in the glucose."

The work of Spies and his co-workers[19, 20] on Vitamin B_6 has been most significant. The application of this knowledge will greatly benefit many cases of the B complex group deficiencies.

I call your attention to the publication "The Role of Nutritional Deficiency in Nervous and Mental Disease," a report of the Association for Research in Nervous and Mental Disease, proceedings of the Association 1941 meeting,[21] and especially those articles by Russell Wilder, W. H. Sebrell, Tom D. Spies, et al, Karl Bowman

and Herman Wortis and James M. McLester. This treatise is highly instructive from the first to the last page.

Berry[22] reporting gastroscopic studies on 100 persons of unquestionable chronic alcoholism gave 30 per cent showing no gastritis, 35 per cent showing a mild chronic superficial gastritis and 35 per cent to have an unequivocal chronic gastritis. My observations indicate that these figures are substantially correct.

Isaac J. Silverman[23] reports on the use of hypertonic saline solution in the treatment of the alcoholic psychosis with very good results. He mentions this treatment in the psychosis of alcoholic pellagrins and these responded nicely, even though the vitamins were withheld, but used later on in the rehabilitation.

In this group of 2275 cases I have not seen one case of delirium tremens. I have seen many cases of alcoholic hallucinosis and many cases of alcoholic or toxic psychosis but not one case of delirium tremens.

In hallucinosis and psychosis vitamins in large doses, along with hypertonic 50 per cent glucose are used immediately. I ocsionally use 5 units of insulin at the same time, never over 10 units of insulin. I prefer the B complex preparations and reinforce them with the thiamin or nicotinamide. This is given intravenously once in twenty-four hours but the glucose and insulin can be repeated in four to six hours. Vitamins by mouth are also given in large doses, as well as a high proteid diet and one that is readily assimilable and one that the patient will take readily, such as egg-milk, with two or three eggs well beaten and flavored with vanilla given every two or three hours. Protein foods are given freely and insisted on. Magnesium sulphate in large doses acts very well and in my opinion has a definite place in the treatment of these cases.

On two occasions I have used the saline solution as suggested by Silverman and found the results satisfactory where the vitamin and insulin method had produced an improvement but had not cleared the condition of hallucinosis. This was really a combination of the two methods.

Vitamin C has been reported as being lowered in the blood and spinal fluid of these alcoholic psychoses. I have not seen any appreciable effect on the cases of large doses, either intravenously, subcutaneously or orally.

Pellagra is seen frequently, beriberi more often than one would suspect, and moderately severe secondary anemias are not infrequent in the alcoholic patient and are not a result of the alcohol per se but a result of the state of malnutrition resulting therefrom.

Late investigations and studies on alcoholic cirrhosis are proving conclusively that these hepatic changes are due to a deficiency disease and most probably the B complex group associated with a low protein intake. The early diagnosis of liver damage has been greatly facilitated by improved laboratory technics, and therefore, early treatment is possible. The use of methionine and choline and proper diets and other therapeutic measures will prevent many of the cases of alcoholic hepatitis from progressing to a cirrhosis.

I would call attention to a severe acidosis that does occasionally develop as a result of the starvation. Hartmans solution acts well in these cases and rarely does it have to be repeated. Dextrose 5 per cent with sodium lactate has also been used with good results.

Advancement in the treatment of the alcoholic during the past decade has been in great part due to advancement in our knowledge of nutrition. Every advance by the nutritional specialist or research worker in this field adds another something to our armamentarium in treating the alcoholic. He is a nutritional guinea pig of his own making. It remains for psychiatrists to apply the knowledge passed on to us by the nutritional experts to help the alcoholic to regain his health.

This writer believes from clinical observation that a powerful narcotic, such as alcohol, when used excessively, will produce an addiction of varying intensity in the sus-

ceptible patient. The various addiction diseases deserve special studies along this line. Basically there is no difference between the opium group addicts and the alcoholic addict. The mechanism of addiction is the same and the abrupt withdrawal symptoms may be very stormy in both types. The craving or desire can be very strong in the alcoholic. The addiction phase of these cases has not been stressed sufficiently.

A great deal has been said and written about will power or volition. My opinion is that it is only good or strong when the nervous system has regained its stability; therefore, the alcoholic is not in a position to remain well until his nervous and emotional stability has been restored and the proper insight given by psychiatry.

I would call your attention to the fact that alcoholism may be a result of organic brain disease. We all know that this is a frequent fact in neurosyphilis. This writer in 1935[24] reported alcoholism as a symptom of Von Economo's encephalitis. In this article I am presenting in tabulated form the spinal fluid findings in 15 cases with characteristic cell-globulin dissociation, increased globulin, negative colloidal gold, and Wasserman. This is true in all cases except one and in this particular case the cells were elevated and changes were found in the colloidal gold test. The clinical symptoms were mostly in the emotional sphere plus alcoholism. It is my opinion that alcoholism uncomplicated by any inflammatory brain disease does not show spinal fluid changes. This viewpoint holds for avitaminosis. Kaplan[25] states that the serology of chronic alcoholism is negative. Alpers[26] also states: "The spinal fluid is normal in chronic alcoholism." Merrit and Fremont-Smith[27] state: "The cerebronspinal fluid is usually entirely normal in cases of polyneuritis associated with acute and chronic alcoholism." These cases are presented for your consideration. The author thinks they are chronic types of Von Economo's encephalitis with alcoholism as a symptom.

The neurological manifestations of the peripheral nervous system are mostly covered by the neuritides. Vitamin therapy is the treatment par execellence but in the acute phase the infra-red ray does help. Where reaction of partial degeneration has set in the addition of the galvanic, sinusoidal and faradic currents is of great value and sometimes surprisingly so.

SPINAL FLUID ANALYSIS*

	CELL COUNT	GLOBULIN TEST	NOGUCHI TEST	PANDY TEST	SUGAR MGS. PER 100cc	TOTAL PROTEID MGS. 100cc	GOLD COLLOIDAL	MODIFIED KOLMER WASS.
A. S. H.	1	Positive	†††	†††	60	35 mgs.	(0000000000)	Negative
C. S. A.	1	Positive	†††	†††	64	32 mgs.	(0000000000)	Negative
L. D. K.	1	Positive	††††	††††	74	50 mgs.	(0000000000)	Negative
M. B.	0	Positive	†††	†††	76	30 mgs.	(0000000000)	Negative
M. B. D.	3.1	Positive	†††	†††	56	30 mgs.	(0000000000)	Negative
J. E.	55	Positive	††††	††††	50	40 mgs.	2234432100	Negative
T. R. W.	1	Positive	†††	†††	40	30 mgs.	(0000000000)	Negative
J. G.	1	Positive	†††	†††	68	30 mgs.	(0000000000)	Negative
C. B. M.	1	Positive	†††	†††	55	25 mgs.	(0000000000)	Negative
H. P.	1	Positive	†††	††††	53	30 mgs.	(0000000000)	Negative
M. S.	1	Positive	††††	††††	71	42 mgs.	(0000000000)	Negative
T. P.	0	Positive	†††	††††	70	25 mgs.	(0000000000)	Negative
B. S.	3	Positive	††††	††††	70	56 mgs.	1222220000	Negative
W. W. B.	0	Positive	††††	††	55	55 mgs.	(0000000000)	Negative
C. L.	0.3	Positive	††††	††††	66	38 mgs.	(0000000000)	Negative

* Dr. W. H. Harris and Dr. A. V. Friedrichs, Clinical Pathologists
Maison Blanche Building
New Orleans, La.

CONCLUSIONS

1) Every alcoholic has a psychiatric problem. This psychiatric problem varies in intensity with each case.

2) The nutritional phase is most important and improvement in this phase is often paralleled by an improvement in psychiatric symptoms.

3) Every alcoholic is an addict.

4) The vast majority of these cases should be institutionalized for study and treatment.

5) Every alcoholic given the proper help can get well and stay well, provided he accepts the fact that under all circumstances he cannot take an alcoholic drink of any type.

BIBLIOGRAPHY

1. Goodhart, Robert, Jolliffe, Norman: Effects of vitamin B_1 therapy on the polyneuritis of alcohol addicts, J. A. M. A. 110:414-419, Feb. 5, 1938.

2. Jolliffe, Colbert and Joffe: Observations on the etiologic relationship of vitamin B_1 to polyneuritis in the alcohol addict, Am. J. Med. Sc. pages 191-515, April 1936.

3. Piker, Philip, Cohn, Jess V.: The comprehensive management of delirium tremens, J. A. M. A. 108:345, January 30, 1937.

4. Spies, Tom D., Chinn, Austin B., McLester, James B.: Severe endemic pellagra, J. A. M. A., 108:853, March 13, 1937.

5. Spies, Tom D., Cooper, Clark, Blankenhorn, M. A.: The use of nicotinic acid in the treatment of pellagra, J. A. M. A., 110:622, February 26, 1938.

6. Askey, J. M., California and Western Med., 51:294, November 1939.

7. Jolliffe, N., Bowman, K., Rosenblum, L. A. and Fein, H. D.: Nicotinic acid deficiency encephalopathy, J. A. M. A., 114:307, January 27, 1940.

8. Report on the Third International Congress on Nutrition, J. A. M. A. Foreign Letters, 114:426, February 3, 1940.

9. Spies, Tom D., Vilter, Richard W.: South. M. J., 33:663, June 1940.

10. Kiene, E., Streitweiser, R. J., and Miller, H.: The role of vitamin B_1 in delirium tremens, J. A. M. A., 114: 2191, June 1, 1940.

11. Robinson, G. W., Jr., and Shelton, P., Kansas City, Mo., Journal-Lancet, 60:433-478, October 1940.

12. Editorial: J. A. M. A., 115:1890, November 30, 1940.

13. Jolliffe, Norman: Vitamin deficiencies and liver cirrhosis in alcoholism, Quarterly Studies on Alcohol, Pages 1-517, December 1940.

14. Vitamins in chronic alcoholism, J. A. M. A., Queries and Minor Notes, 114:2491, June 22, 1941.

15. Alexander, Leo: Quarterly Journal Studies on Alcohol, pages 11-260, September 1941.

16. Bean, William Bennett, Spies, Tom D., Blankenhorn, Marion A.: The incidence of pellagra in Ohio hospitals, J. A. M. A., 118:1177, April 4, 1942.

17. Strecker, Edward A., Rivers, Thurston D.: Adjuvant treatment of toxic states; Preliminary report, Philadelphia Psychiatric Soc. Arch., Neurol. & Psych., 47:697, 1942.

18. Cannon, E. A., Modarelli, W. H., De Vincenzo, F. R., Swiney, M., III., The treatment of delirium tremens, J. A. M. A., 119:1418, August 22, 1942.

19. Spies, T. D., Bean, W. B. and Ashe, W. F.: A note on the use of vitamin B_6 in human nutrition, J. A. M. A., 112:2414, June 10, 1939.

20. Spies, T. D., Landisch, R. K., and Bean, W. B.: Vitamin B_6 (Pyridoxin) deficiency in human beings, J. A. M. A., 115:839-840, September 7, 1940.

21. The Role of Nutritional Deficiency in Nervous and Mental Disease. The Williams and Wilkins Company, Baltimore 1943. Research Publication Association Nervous and Mental Research.

22. Berry, L. H.: J. A. M. A., 117:2333, December 27, 1941.

23. Silverman, Isaac J.: Med. Ann. Dist. Col., 13:102.

24. Young, Roy Carl: Chronic epidemic encephalitis: report of eleven cases with chronic alcoholism as the outstanding symptom, New Orleans M. and S. J., 88:150-158, September 1935.

25. Kaplan, D. M.: Serology of Nervous and Mental Diseases, W. B. Saunders and Co., page 124, 1914.

26. Alpers, Bernard: The Human Cerebrospinal Fluid, Association for Research in Nervous and Mental Diseases, Publication Paul Hoeber, Inc., New York, page 444, 1924.

27. Merritt and Fremont Smith: The Cerebrospinal Fluid, W. B. Saunders and Company, Philadelphia, page 181, 1937.

—o—

MYXEDEMA
A REPORT OF TEN CASES FROM A GENERAL HOSPITAL

EDWIN F. CHANTON, M. D.*

AND

MARION D. HARGROVE, M. D.†

SHREVEPORT

Hypothyroidism is a term referring to any state in which an inadequate amount of thyroid hormone is made available to the tissues. This inadequate supply of thyroid hormone may be temporary or permanent, and to a varying degree of severity. If the hypothyroidism is of sufficient degree and duration, clinical alterations appear which establish a clinical entity.

There are several types of thyroid insufficiency, which as clinical entities are in part dependent on age and in part dependent upon the means by which the thyroid insufficiency was produced. These are spontaneous adult myxedema, myxedema following thyroiditis, postoperative myxedema, and endemic myxedema or cretinism. These first four are believed to be essential results of deficient thyroid function, while endemic myxedema is believed to be a more complex disorder in which other glands in addition to the thyroid are involved.[1]

We will deal here with spontaneous adult

*Assistant Resident in Medicine, Charity Hospital at Shreveport, La.

†Chief Visiting Physician, Charity Hospital at Shreveport, La.

myxedema and shall henceforth refer to it as "Myxedema".

Myxedema develops slowly and usually in patients with no previous history of thyroid disease. It occurs most frequently in females of middle life but may occur in males and other age groups. Pathologically, the only constant finding in both treated and untreated cases is atrophy and virtual absence of the thyroid gland.[2]

The onset is relatively insidious and first manifested by complaints of weakness, easy fatigability and sensitivity to cold. A gain in body weight is commonly found but sometimes the appetite is so poor that this does not occur. In time there follow the characteristic signs of puffiness of the eyes, dryness of the skin, a tendency for the hair to fall out and the almost complete disappearance of sweating. Menorrhagia is common but may be absent. Over a period of years the clinical condition progresses and puffiness of the tissues over the entire body appears. Speech becomes thick and the voice hoarse and deep with a low pitch. Somnolence increases and there is a marked slowness in responding to questioning. Eventually imbecility may intervene.

Laboratory examinations will usually show a high blood cholesterol; and an anemia, either of the hypochromic or macrocytic type, is commonly found. Hypochlorhydria or achlorhydria will be found in the majority of cases.[4] The urine may contain albumen and the phenolsulfonphthalein test gives low results but the renal function is not significantly impaired.[5] The basal metabolic rate will be low.

A particular form of heart disease is a part of the clinical picture of advanced myxedema. It is characterized by cardiac enlargement, a slow pulse rate, and reduced cardiac output.[2] The blood pressure is usually low but in the later stages of the disease may be elevated.[8] Electrocardiographic changes are present which consist of flat or inverted T waves, small P waves, and QRS complexes of low amplitude. The heart shows moderate enlargement on x-ray. Pathologically at postmortem, the myxedemic heart will show changes which are characteristic of this condition but not specific for it.

La Due[7] describes three cases of myxedema which died untreated. In all three cases the heart was grossly dilated, the myocardium pale, and narrowing of the coronary arteries with atheromatous changes was found. Section of the heart muscle showed replacement of the sarcoplasm of the myofibrils by hydropic vacuoles in many places. Some of the cells were pale while others were deeply stained and had pyknotic nuclei. Stains of the vacuoles were negative for fat, mucus, and glycogen, indicating a true hydroscopic swelling similar to that noticed in the edematous tissues of patients with myxedema. Clinically the size of the heart and EKG findings revert to normal under thyroid therapy. The heart rate increases as does the stroke volume and if any failure is manifested originally, it disappears.

The thyroid hormone is not necessary to life since the oxidative processes of the body continue in its absence. However, in man the resting rate of oxygen consumption is reduced one-third to one-half in total absence of the thyroid hormone.

There is found a reduced rate of protein metabolism in hypothyroidism with storage of a considerable amount of protein in the interstitial fluid. The peculiar edema of hypothyroidism is thought to be due to a progressive deposit of protein in the interstitial spaces, which tends to withdraw fluid from the plasma with a resultant increase in the volume of interstitial fluid, a decrease in the volume of plasma, and an increase in the concentration of plasma protein. According to Boothy[6], this consists of a semifluid albuminous substance containing about 13 per cent protein and represents an increase in the normal quantity of protein. With administration of thyroid, a diuresis takes place and a considerable portion of this protein undergoes metabolism and is excreted in the urine. However, no specific diuretic effect of the thyroid hormone can be demonstrated.

Both free and esterified cholesterol are elevated in hypothyroidism. Green[10], in

a series of cases at the Lahey Clinic, reported nine patients with an average serum cholesterol level of 399 milligrams per cent before treatment with thyroid extract. These patients were given thyroid therapy and the serum cholesterol at the termination of study (when the patients were symptom-free) averaged from 120 to 250 milligrams per cent with a mean average of 180 milligrams per cent. There are exceptions to the rule and numerous cases of myxedema have been seen with apparently normal cholesterol levels. Peters and Mann[2] explain this by stating that some individuals normally tend to have high cholesterol values while others tend to have low values. With thyroidectomy or thyroid atrophy, these values will rise to the same extent in both individuals. However, in those individuals whose cholesterol level was initially high the rise will be accentuated, while in those individuals whose initial cholesterol level was low the rise will be partially masked. Nutrition and food intake also have a direct bearing on the cholesterol blood level and may distort and mask the picture. In cases of malnutrition, poor appetite, or any poor food intake, in spite of a hpyothyroid condition the cholesteral level will be low. But, if the patient is fed by tube or given an adequate diet, the cholesterol level will rise proportionately. Steiner and Domenski[9] fed ten patients on high cholesterol diets for eight to ten weeks. The serum cholesterol in each increased from 50 to 218 milligrams per cent with an average rise of 101 milligrams per cent.[9] High cholesterol values are also found in diabetes mellitus, lipoid nephrosis, certain types of liver disease and obesity.[11]

Diagnosis of myxedema can only be made on the proper evaluation of clinical signs and symptoms, together with BMR and serum cholesterol values. It is important, however, to evaluate the case as a whole and not place particular emphasis or reliance upon a single procedure. The specific laboratory means of diagnosing early myxedema is the serum precipitable iodine level which is depressed in this condition and in no other as shown by Bassett, Coons and Salter.[12]

The myxedematous patient, especially if the condition has been severe and of long standing, exhibits a peculiar initial hypersensitivity to thyroid extract. This is even more apt to occur in the severe cases with cardiac manifestations.[3] Means[13] states emphatically that in inaugurating thyroid therapy the physician should proceed with extreme caution and have the patient under surveillance. Thyroid extract is a cumulative drug, the full effects of a single dose being delayed for ten days.[14] The effects of the drug may last as long as seventy days. However, we may assume that therapeutic effects will have passed off in about thirty days.

When giving thyroid it is well to give it by the cumulative plan. This involves giving a small amount at the beginning, at such intervals so as to build up a concentration in the body adequate to produce a therapeutic effect. For proper evaluation of the dosage we must know when the peak effect of a single dose is reached and when the maximum therapeutic level is attained. Goodman and Gilman[14] state that the final decision regarding a given dose level of thyroid cannot be reached until the patient has been treated for several weeks. The

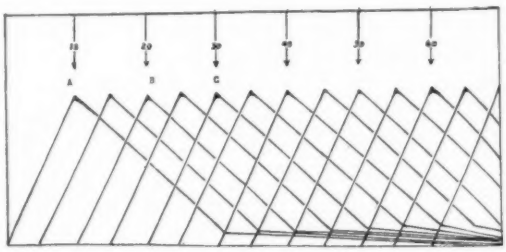


Fig. I

Legend: Schematic graph illustrating the cumulative effect of repeated single doses of thyroid extract; letter A represents the time interval required for the maximum effect of a single dose of thyroid extract; letter B represents the time required from onset of dosage to obtain the maximum cumulative therapeutic effect; letter C represents the point of elimination of the first single dose of thyroid extract. The time interval is represented in ten day periods by arrows at the top of the chart.

following graph will clearly illustrate this point. Thus the full effect is obtained about the third week when we have summation of the waning effect of the first dose plus the various stages of effect of other single doses.

It has been found that overdosage or too rapid dosage of thyroid extract in cases of long standing myxedema may produce serious results. Anginal attacks have been reported as well as arrythmias and death.[2, 8] Aisner[16] reports a case of complete heart block resulting from excessive dosage of thyroid extract, while cases of bundle branch block and auricular fibrillation have been also reported.[17]

We here report two cases in which we feel the cause of death was too vigorous thyroid medication. We believe that in advanced cases of myxedema thyroid dosage should be started slowly and in small amounts, bearing in mind the fact that the cumulative effect of the drug will not be fully observed for three weeks. Duncan[2] recommends beginning dosage of half a grain a day for three or four days, then discontinuance of therapy for several days after which the patient should be continued on the half-grain dose. The maintenance dosage of thyroid extract should be estimated by observation of the patient and regression of symptomatology, not the basal metabolic rate or cholesterol level. The patient, accustomed to a slow way of life may feel much better when the basal metabolic rate is about minus 10 than she would if it were at zero and her metabolism more rapid.[2, 8, 18] Once on a comfortable maintenance dose the patient may be carried on indefinitely with no return of symptomatology. Burgess reports a case which has been maintained comfortably on thyroid extract for fifty-two years.

CASE REPORTS

Case No. 1. A forty-eight year old white woman entered the hospital complaining of dyspnea, anorexia and weakness. Her illness dated back seven years when she first noticed dryness of the skin and loss of hair, followed by huskiness of the voice, difficulty in speaking, and a general inability to get things done.

Physical examination revealed an obese, drowsy, middle-aged female. The face was heavy, pallid and expressionless, and there was a moderate amount of periorbital edema present. The voice was husky and speech halting. The skin was dry and scaly and the subcutaneous tissue was thickened but did not pit on pressure. Examination of the heart, lungs, and abdomen revealed no abnormalities. The blood pressure was 215/140.

Laboratory studies revealed a normal blood picture, a serum cholesterol level of 500 milligrams per cent, BMR's of minus 24 and minus 26 and achlorhydria.

The patient was placed on 4 grains of thyroid extract daily on the fifth hospital day. On the ninth hospital day the patient complained of headache. On the eleventh day the headache was more severe and she was nauseated and vomiting. The patient expired on the seventeenth hospital day.

Necropsy revealed marked arteriosclerosis, thyroid atrophy and an enlarged edematous heart. No anatomical cause of death could be found but it was felt that death had resulted from the excessive thyroid medication.

Case No. 2. A seventy year old white female entered the hospital complaining of easy fatigability, gradual gain in weight, and malaise for five years duration.

Physical examination revealed an obese white female with dry scaly skin and a hard non-pitting edema over the entire body. Examination of the heart, lungs, and abdomen revealed no abnormalities. Laboratory studies revealed a mild anemia, a serum cholesterol level of 290 milligrams per cent, and BMR's of minus 27 and minus 23.

The patient was placed on 2 grains of thyroid extract a day and maintained on this dosage for twenty days, at which time she began to evidence signs of toxicity, precordial pains and lethargy. In spite of this the thyroid dosage was raised to 6 grains a day and maintained at this level for fifteen more days. During this time the patient became increasingly toxic and complained of pain in the head and chest. The thyroid was discontinued on the thirty-fifth day after medication was started. A serum cholesterol level taken the day before exitus was minus 59 milligrams per cent.

It was felt that the cause of the patient's death was excessive dosage of thyroid extract.

We feel that the death of both of these patients was directly attributable to excessive dosage of thyroid extract. They were both started on what we consider massive dosage. The first patient received 4 grains of thyroid extract a day which was continued until exitus in spite of the development of the general symptoms of toxicity which should have suggested to the physician the discontinuance of the drug. The second patient first received 2 grains of thyroid extract; however, this dosage

was shortly raised to 6 grains a day. This patient developed definite symptoms of toxicity which were recognized and although thyroid medication was discontinued she had already received enough of this slowly excreted drug to cause her death.

Case No. 3. A twenty-eight year old white female entered the hospital complaining of menorrhagia, generalized weakness, and malaise of one year's duration. She had also noticed a generalized swelling of the body, particularly the face.

Physical examination revealed an obese white female with dry scaly skin and periorbital edema. Examination of the heart, lungs, and abdomen showed no abnormalities. The blood pressure was 80/60. Blood studies revealed a slight anemia. BMR's were minus 41 and minus 46.

The patient was placed on 4 grains of thyroid extract a day on the fifth hosiptal day and maintained on this dosage for twenty-one days, after which she was discharged on 2 grains a day. At this time she showed marked clinical improvement and her BMR was minus 19.

Case No. 4. A thirty-two year old colored female entered the hospital complaining of weakness, menorrhagia, and lassitude of seven years duration. She had also noticed falling out of her hair and puffiness of the face.

Physical examination revealed an obese colored female with a puffy appearing face, dry scaly skin, and coarse scanty hair. Examination of the heart, lungs, and abdomen showed no abnormalities. The blood pressure was 90/60. Laboratory studies revealed a slight anemia, a serum cholesterol level of 300 milligrams per cent, and BMR's of minus 37 and minus 29.

The patient was placed on 1 grain of thyroid extract a day and maintained on this dosage for eleven days. During this time she showed some clinical improvement. However, she signed out of the hospital on the eleventh day and no follow-up could be made.

Case No. 5. A fifty-one year old white female entered the hospital complaining of generalized swelling, weakness, and easy fatigability of seven years' duration.

Physical examination revealed an obese white female with thickened features, puffy face, and dry scaly skin. The voice was hoarse and speech was halting. Examination of the heart, lungs, and abdomen showed no abnormalities. Laboratory studies revealed a slight anemia, a serum cholesterol level of 260 milligrams per cent, and BMR's of minus 37 and minus 36.

The patient was placed on ½ grain of thyroid extract a day on the fifth hospital day. This dose was raised gradually half a grain at a time until on the thirtieth hospital day the patient was receiving 2 grains a day. The patient's course in the hospital was excellent, and on the forty-fifth day she was discharged much improved.

Case No. 6. A forty-four year old colored female entered the hospital complaining of progressive weakness and sensitivity to cold. She had noticed that her voice was becoming increasingly hoarse and that her face was swelling.

Physical examination revealed a lethargic colored female with dry scaly skin and sparsity of hair. The voice was hoarse and speech halting. A hard non-pitting edema was present over the entire body. The blood pressure was 108/80. Examination of the heart, lungs, and abdomen showed no abnormalities. Laboratory studies revealed a slight anemia, a serum cholesterol level of 292 milligrams per cent, and BMR's of minus 31 and minus 33.

The patient was started on thyroid extract, 1½ grains a day. The patient had an excellent hospital course and was discharged after thirty days on the same dosage of thyroid extract.

Case No. 7. A sixty year old white female entered the hospital complaining of a gradual increase in weight over the last three years accompanied by puffiness of the face and increasing hoarseness.

Physical examination revealed an obese white female with coarse hair, a dry scaly skin and puffy face. The voice was hoarse and speech halting. Examination of the heart, lungs, and abdomen showed no abnormalities. The blood pressure was 120/75. The serum cholesterol was 427 milligrams per cent and the BMR's were minus 27 and minus 23. The electrocardiograms showed a diphasic T1 and T4, low amplitude T2 and T3, and low voltage QRS complexes in all leads.

The patient was placed on 1 grain of thyroid extract a day on the second hospital day. On discharge sixty days later the patient felt much better and symptomatology had completely regressed.

Case No. 8. A fifty year old white female entered the hospital complaining of gradually increasing weakness, fatigability and sensitivity to cold.

Physical examination revealed an obese white female with puffy face, dry scaly skin and sparse hair. The voice was deep and hoarse and speech halting. Examination of the heart, lungs, and abdomen showed no abnormalities. The blood pressure was 90/60. Laboratory studies revealed a mild anemia, serum cholesterol levels of 325, and 312 milligrams per cent, and BMR's of minus 17.

The patient was placed on thyroid extract, 1 grain daily. Her course in the hospital was excellent and she was discharged in nineteen days, showing much symptomatic relief.

Case No. 9. A sixty-six year old white female entered the hospital complaining of gradual increasing weakness, fatigability and sensitivity to cold.

Physical examination showed an obese white female with edematous face, dry, scaly skin, sparse

hair and coarse features. The voice was hoarse and speech was halting. Examination of the heart, lungs, and abdomen showed no abnormalities. The blood pressure was 180/104. Laboratory studies revealed a serum cholesterol level of 506 milligrams per cent and a BMR of minus 24. The electrocardiogram showed inverted T waves and low amplitude QRS complexes in all four leads.

Thyroid extract, 1 grain a day, was started and continued throughout the patient's course in the hospital. The patient's recovery was excellent and she was discharged after forty-five days with complete regression of symptoms.

Case No. 10. A forty-eight year old white female entered the hospital complaining of progressive weakness, fatigability and sensitivity to cold. She also complained of gain in weight and swelling of the face.

Physical examination revealed a lethargic, obese white female with coarse features, facial edema and dry scaly skin. The voice was hoarse and speech halting. Examination of the heart, lungs, and abdomen showed no abnormalities. The blood pressure was 130/90. Laboratory studies revealed a slight anemia, serum cholesterol levels of 211 and 244 milligrams per cent, BMR's of minus 23, minus 33 and minus 33, achlorhydria, 35 per cent P. S. P. excretion and urine concentration of 1.025. The electrocardiogram showed inverted T waves and low amplitude QRS complexes in all four standard leads. X-ray of the chest showed slight generalized cardiac enlargement.

The patient was placed on half a grain of thyroid extract a day for twelve days after which the dose was raised to a grain a day. The patient was observed in the hospital while on this dosage for three weeks, and then discharged on a grain a day maintenance dose. Her course in the hospital was excellent and she left after complete regression of symptoms although her BMR was still minus 17.

CONCLUSIONS

In the study of 126,000 admissions at Shreveport Charity Hospital in the last nine years, 10 cases of myxedema were found. These occurred entirely in females in the middle years of life, and were confined in a majority of cases to the white race.

Thyroid extract has a cumulative effect, the full therapeutic value of the drug is not reached until at least three weeks after the start of administration and the drug is not fully excreted until at least three weeks after being discontinued.

Thyroid extract when given in excessive dosage may cause cardiac complications and death.

BIBLIOGRAPHY

1. Tice: Practice of Medicine, Vol. 16.
2. Winkler, A. W.: Diseases of the Thyroid Gland, Disease of Metabolism, Duncan: 896.
3. Greene, A. M.: Iodine and cholesterol metabolism in patients with primary myxedema, Arch. Int. Med., 67:114, 1941.
4. Golding, F. C.: The association of atrophic gastritis with hypothyroidism, Ann. Int. Med., 22:828, 1942.
5. DuBois, E. F.: Myxedema, Cecil, Textbook of Medicine, Sixth Edition, 1216.
6. Best and Taylor: Physiological Basis of Medical Practice, Third Edition, 1125.
7. LaDue, J. S.: Myxedema Heart, Ann. Int. Med., 18:332, 1943.
8. Barr, D. P.: Diseases of the Thyroid Gland, Cecil, Textbook of Medicine, Seventh Edition, 1334.
9. Steiner, A.: Dietary hypercholesterolemia, Am. J. Med. Sc., 201:820, 1941.
10. Green, A. M.: Arch. Int. Med., 67:144, 1941.
11. Stone, C. T.: The clinical importance of cholesterol, South. M. J., 28:705, 1935.
12. Means, J. H.: Some new approaches to the physiology of the thyroid, Ann. Int. Med., 19:500, 1943.
13. Means, J. H.: Diseases of the thyroid gland, New England J. M., 231:820, 1939.
14. Goodman, L. and Gillman: The Pharmacological Basis of Therapeutics, 1166.
15. Gold, H.: Conference on Therapy ,Am. J. M., page 296, March 1947.
16. Aisner, M.: Complete heart block resulting from overdosage with thyroid extract, New England J. M., 221: 336, 1939.
17. Rodgers, H. M.: The cardiovascular manifestations of induced thyrotoxicosis, Ann. Int. Med., 26:914, 1947.
18. Burgess, A. M.: Myxedema controlled by thyroid extract for 52 years, Ann. Int. Med., 25:146, 1946.

—o—

HUMAN EQUINE ENCEPHALOMYELITIS, EASTERN TYPE, IN LOUISIANA

GEORGE H. HAUSER, M. D.*

NEW ORLEANS

The first human case of equine encephalomyelitis in Louisiana, due to the eastern type virus was reported in 1946,[1] and in 1947 there were ten proven cases. This disease has been reported only in the United States within the last decade. The first outbreak occurred in Massachusetts in 1938,[2] and Fothergill and associates,[3] and Webster and Wright,[4] isolated the eastern type of equine encephalomyelitis virus from the brain tissue of children dying of the disease, thereby establishing the etiology. In the following year, McAdams and

Read before the Sixty-eighth Annual Meeting of the Louisiana State Medical Society, Monroe, April 14, 1948.

*Director, Division of Laboratories, Louisiana State Department of Health, New Orleans, La.

Porter[5] isolated the virus from the brain of an adult who succumbed to the disease. Since the Massachusetts outbreak, the only other cases reported, except those from Louisiana, were three cases from Texas in 1942,[6, 7] and three in 1947,[8, 9] all confirmed by the presence of neutralizing anitbodies in the blood.

On September 8, 1947, the Louisiana State Department of Health was notified that two children from the same family residing in Southwest Louisiana had been admitted to New Orleans Charity Hospital where a tentative diagnosis of encephalomyelitis had been made. Members of the Health Department investigated the cases and found that both children were acutely ill with symptoms typical of encephalomyelitis. The following day one of the children died. Autopsy was performed and part of the brain and cord tissues were frozen, part placed in buffered glycerol solution, and sections of all tissue taken for microscopic examination. The eastern type virus was isolated from the brain tissue.

Three days later, the other child expired and by this time several other suspected cases had been admitted to the Charity Hospital in New Orleans and the Charity Hospital in Lafayette, La. At the request of the State Health Officer, the United States Public Health Service sent an epidemiologist, and a veterinary pathologist, to aid in the investigation. Accompanied by an entomologist of the Health Department this so called "encephalomyelitis team" visited the affected area, investigated some of the cases and collected insects, and specimens of blood from contacts and various animals. The results of these studies will be published by them later.

During 1947, the Health Department has had the opportunity to study 32 cases which were reported as suspected of having equine encephalomyelitis. Of these, 22 were found to be due to other diseases such as tuberculosis, meningitis, tonsillitis, mumps, etc., and 10 definitely diagnosed as equine encephalomyelitis, eastern type, (Table 1). The eastern type virus was isolated from the brain tissue of 2 cases, 7 showed positive neutralizing antibody tests, 1 positive complement fixation test, and 7 showed pathognomonic lesions in the brain tissue on microscopic examination. (Table 2) Of the 10 cases, 7 died and 3 recovered, the case fatality rate being 70 per cent.

The age distribution of cases which occurred in Louisiana substantiates the theory that children are more susceptible than adults. All, except one, were in children, from 7 months to 15 years of age. An unusual case in the series was that of a colored male, age 74 years, who developed severe symptoms and died wtihin five days after onset of the disease. The cases were rather evenly distributed among the sexes, 6 occurring in males, and 4 in females.

TABLE 1
SUMMARY OF HUMAN CASES

Patient	Address	Age	Sex	Color	Date of Onset	Date of Death	Date of Discharge
						9-12-47	
H DeM.	Maurice, La.	5	M	W	9- 2-47	9- 9-47	
E. DeM.	Maurice, La.	2	F	W	9- 4-47	9-17-47	
J. D.	Delcambre, La.	74	M	W	9-12-47		
F. J.	Weeks Isle, La.	15	F	C	9-16-47		10-15-47
E. DeR.	Weeks Isle, La.	11	F	W	9-17-47		10-19-47
M. I. S.	Franklin, La.	7 Mo.	M	C	9-23-47	11-16-47	
A. L.	Bay St. Louis, Miss.	3	F	W	10- 4-47	10-28-47	
L. S.	Cut Off, La.	4	M	W	10- 9-47	10-13-47	
M. R.	Gibson, La.	5	M	W	10-19-47	10-21-47	
A. B.	Theriot, La.	5	M	W	10-23-47		12-11-47

TABLE 2
EQUINE ENCEPHALOMYELITIS, EASTERN TYPE VIRUS
DIAGNOSTIC FINDINGS

Patient	Virus Isolation (Brain	Neutralizing Antibodies (Blood)	Complement Fixation Test (Blood)	Microscopic Pathology
H. DeM.	Negative *2, 3	WEAKLY POSITIVE *3	Negative *4	POSITIVE *5, 6
E. DeM.	POSITIVE *1, 2, 3 (Eastern Type)	WEAKLY POSITIVE *3	Negative *4	POSITIVE *5, 6
J. D.	POSITIVE *2, 3 (Eastern Type)	Negative *2, 3	Negative *4	POSITIVE *5
F. J.		POSITIVE *2, 3	POSITIVE *	
E. DeR.		POSITIVE *3	Negative *4	
M. I. S.	Negative *2, 3	POSITIVE *3	Negative *4	POSITIVE *5, 6
A. L.	Negative *2, 3	Negative *3	Negative *4	POSITIVE *5, 6
L. S.	Negative *2, 3	POSITIVE *3	Negative *4	POSITIVE *5, 6
M. R.	Negative *2, 3	Negative *3	Negative *4	POSITIVE *5, 6
A. B.		POSITIVE *2, 3		

*Laboratories performing tests:
1. Donaldson, P., and Shaffer, M. S., Tulane University, School of Medicine, New Orleans, La.
2. Dascomb, H. E., and Syverton, J. T., L. S. U. School of Medicine, New Orleans, La.
3. Howitt, B. F., U. S. P. H. S., Virus Laboratory, Montgomery, Ala.
4. Hammon, W. McD., Hooper Foundation, San Francisco, Calif.
5. Dent, J., Tulane University, School of Medicine, New Orleans, La.
6. Hauser, G. H., La. State Department of Health, New Orleans, La.

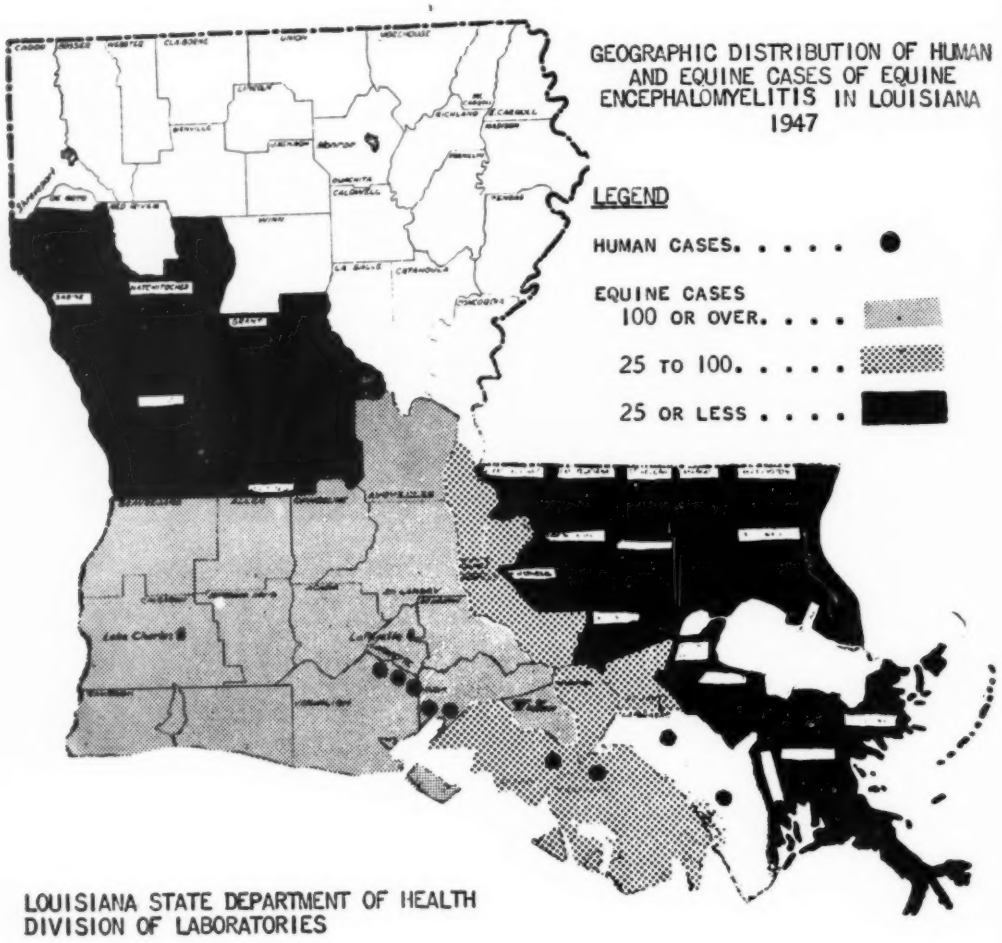
TABLE 3
HUMAN CASES
AGE AND SEX

Age	Male	Female	Total	Percentage of Total
Under 1	1	0	1	10
1-4	1	2	3	30
5-9	3	0	3	30
10-15	0	2	2	20
74	1	0	1	10
Totals	6	4	10	100

The first case was reported on September 8, 1947, and the last case on October 23, 1947. The disease appears to occur characteristically in the late summer and autumn. Nine cases reported in January and February, 1948, as suspicious of equine encephalomyelitis, have proved negative to date. In a field study, of the area from which these cases originated, only three mosquitoes and one hog louse were collected, probably due to severe cold weather. As all suspected cases and the areas from which they originate are investigated by the State Department of Health, it is imperative that physicians throughout the state report any suspected case.

Figure 1 shows the geographic distribution of human cases of equine encephalomyelitis which occurred during the 1947 outbreak. It will be noted that most of the human cases occurred in rural areas or small towns. The map also shows the relation of human cases to equine cases, based on statistics as collected by Piercy[10] from a questionnaire sent to all practicing veterinarians in the state. Accurate figures on the incidence of the disease in animals vary widely, and those of Piercy are very much higher than those of the U. S. Department of Agriculture. It appears that many animal cases are never reported.

As the Massachusetts and Louisiana outbreaks are the only two known to be caused by the eastern type virus, it is interesting to compare our findings with those reported by Feemster[2] and Getting.[11] Statistically, they were quite similar. The distribution among the sexes was rather even and children were particularly susceptible in both


GEOGRAPHIC DISTRIBUTION OF HUMAN AND EQUINE CASES OF EQUINE ENCEPHALOMYELITIS IN LOUISIANA 1947
LEGEND
HUMAN CASES. . . . .
EQUINE CASES
100 OR OVER. . . .
25 TO 100. . . . .
25 OR LESS . . . .
LOUISIANA STATE DEPARTMENT OF HEALTH
DIVISION OF LABORATORIES

of the outbreaks. Seventy per cent of our cases and 70 per cent of the Massachusetts cases were in children under ten years of age. The case fatality rate in Louisiana, in humans, was 70 per cent, while in Massachusetts it was 74 per cent.

Two of the fatal cases in the Louisiana outbreak were from the same household and two other members from the family showed neutralizing antibodies in their blood. This may be of epidemiological significance as multiple cases in the same household were not present in the Massachusetts outbreak. This also raises a possibility of contact infection or vector transmission of the disease from one human to another, although laboratory experiments by various workers have failed to show transmission of the disease by direct contact.

A summary of the clinical symptoms shows that in most cases the onset was sudden with anorexia, irritability, vomiting, sometimes diarrhea, headache, fever, drowsiness and restlessness followed by muscular twitchings, convulsions, and coma. There was rigidity of the neck and stiffness of the back, lower, and upper extremities. Most of the cases were admitted to the hospital from two to five days after onset of illness. Temperature usually ranged from 102° to 106.4° F.

Laboratory findings were rather constant. Blood examination usually showed slight leucocytosis with increase in polymorphonuclears. Blood chemistry (Sugar

and N.P.N.) and urinalysis in most cases were negative. Spinal fluid was clear or slightly hazy in appearance, while pressure, cells, and globulin showed an increase. The polymorphonuclears predominated in the spinal fluid early in the disease, and later the lymphocytes. Blood and spinal fluid cultures were negative for bacterial infection.

In the cases in which recovery took place, symptoms were not as severe, but convalescence was prolonged. One of the distressing features of the disease is that nearly all cases that recover show disabling mental and physical sequelae which are progessive in character. A study by Getting[11] of the 9 surviving patients of the Massachusetts outbreak, approximately one year after onset of illness, showed that 6 suffered disabling sequelae. Feemster,[12] in a nine year study of these cases, reports that not only have these patients shown no further improvement, but that some of the individuals have shown further retardation.

In the fatal cases, the patients grew progressively worse despite chemotherapy and supportive measures, with almost continuous convulsions and coma until death, which usually occurred from two to ten days after onset. One unusual case was that of a 7 month old colored child who died on the fifty-fourth day of illness.

Autopsies were held on the 7 fatal cases. Grossly, the brain and spinal cord showed congestion and marked edema. One case showed areas of hemorrhage. The convolutions of the brain were markedly flattened. Microscopic examination showed more or less similar findings throughout the brain tissue, most marked in the midbrain. These consisted of diffuse infiltration of polymorphonuclear leucocytes, and a lesser number of large mononuclear cells, especially about the blood vessels, in the brain, cord and meninges; small focal areas of polymorphonuclears with abscess formation, destruction of nerve cells, with infiltration of polymorphonuclears and large phagocytic cells (Neuronophagia), many Gitter cells, areas of focal necrosis, proliferation of glial cells, with production of glial nodules, and irregular areas of demyelinization with no relation to the blood vessels. The blood vessels showed proliferation of the lining endothelium, with numerous thrombi in the small vessels, many of which were hyalinized. The walls of the blood vessels showed infiltration of polymorphonuclears with necrosis present. In cases dying after prolonged illness, only a small amount of polymorphonuclear infiltration was present with a greater amount of mononuclear invasion, marked gliosis, atrophy, and cystic changes. Changes in the viscera included congestion, proliferation of the lining of the small vessels, with numerous thrombi, interstitial pneumonitis, and incidental secondary pathological changes.

The diagnosis of encephalomyelitis is usually made clinically and confirmed by laboratory methods. The laboratory procedures of value, to confirm the diagnosis, are: (1) Isolation of the virus, (2) tests for neutralizing antibodies, (3) complement fixation test, and (4) post mortem examination.

1). Isolation of virus: Specimens of blood and spinal fluid should be collected as soon as the disease is suspected, frozen at once, and forwarded to the laboratory for virus isolation.

2). Test for neutralizing antibodies: A specimen of blood should be taken in the early stage of the disease and subsequent specimens at weekly intervals. An increasing titer of neutralizing antibodies during the course of the disease is of great value in confirming the diagnosis.

3). Complement fixation test: This has proved of little value in cases that succumbed after a few days' illness. However, in those that live over two weeks or in those that recover, it may be helpful.

For neutralizing antibody and complement fixation tests, withdraw 10 c.c. of blood by venipuncture, allow to clot, and send to the laboratory at once. If it is to be shipped a long distance, the serum should be separated from the clot.

4). Post mortem examination: In case of death, an autopsy should be performed. It is important that sections of the brain,

and spinal cord be obtained, special care being taken to obtain portions of the midbrain. Part of the specimen should be frozen at once, and the remainder placed in buffered glycerol solution.

It should be forwarded to the laboratory for animal inoculation in an effort to isolate the virus. In cases that have succumbed early, the virus can usually be isolated from the brain tissue; whereas, in cases that have been prolonged, the virus is hard to isolate and appears to have burned itself out. Upon isolation of the virus, the type must be determined by animal protection tests.

Sections of all tissues should also be taken and placed in 10 per cent formalin for microscopic examination.

Complete epidemiology of this disease has not been established. The eastern type virus of equine encephalomyelitis was originally isolated by Ten Broeck and Merrill[13] from the brains of horses dying of the disease along the Atlantic Coast. Transmission of the eastern virus to experimental animals by the bite of infected mosquitos, was first reported by Merrill, Lacaillade and Ten Broeck[14] in 1934. In 1938, Fothergill and Dingle[15] isolated the eastern strain virus from a wild pigeon in Massachusetts, and Tyzzer and associates[16] the virus from two wild pheasants in Connecticut. The same year Ten Broeck[17] found neutralizing antibodies present in the sera of fowls from farms, where horses had died of encephalomyelitis, and the following year (1939)[18] postulated that "birds act as reservoir hosts for the virus." Davis[19] has presented experimental evidence that birds, including English sparrows, pigeons, and cowbirds can be infected with the virus, by the bite of infected mosquitoes.

From all the evidence at hand, it appears that birds are the reservoir for the virus, from which arthropods, principally mosquitoes, become infected and transfer the infection to humans, horses, birds, and other animals.

A mosquito survey made in Massachusetts[20] following the 1938 outbreak showed 21 species of Aedes present, 6 of which had previously been proved to transmit the eastern type virus. In a mosquito survey in Louisiana made by the Louisiana State Department of Health during 1947, 16 species of Aedes were found, 5 of which have been previously proved to transmit the eastern type virus (Table 4), and their seasonal incidence is shown in Figure 2.

TABLE 4

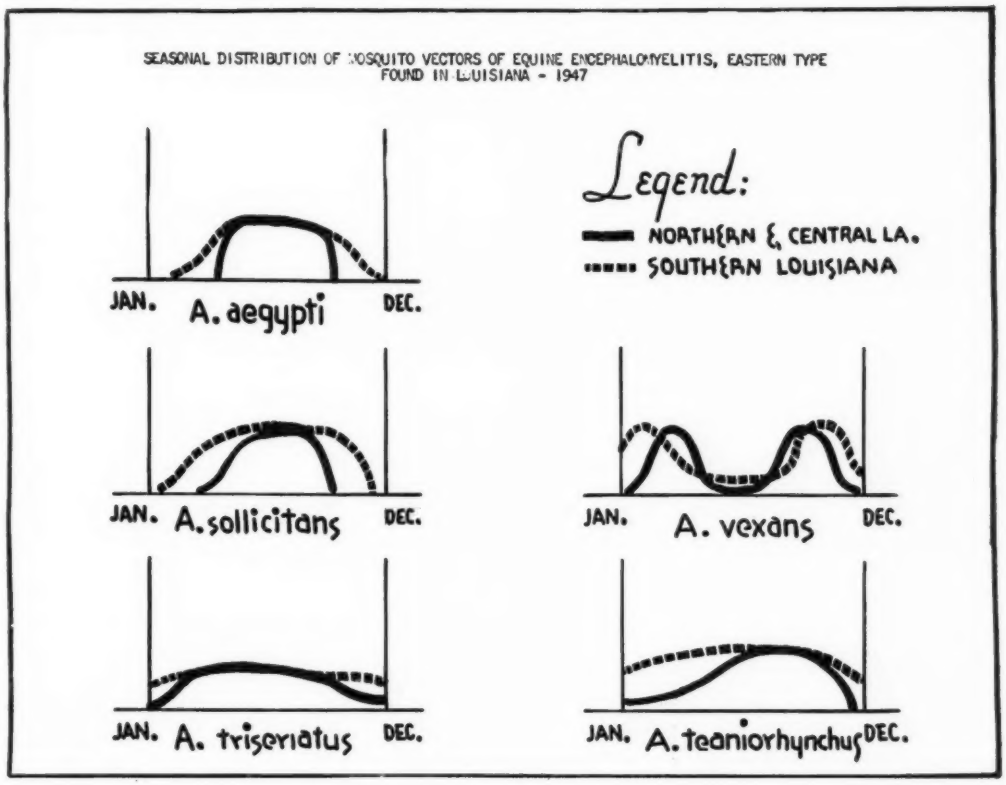
OCCURRENCE OF MOSQUITO VECTORS OF EQUINE ENCEPHALOMYELITIS, EASTERN TYPE, IN MASSACHUSETTS — LOUISIANA

Name of Mosquito	Occurrence in Massachusetts	Occurrence in Louisiana
Aedes aegypti	0	X
A. sollicitans	X	X
A. cantator	X	0
A. vexans	X	X
A. triseriatus	X	X
A. atropalpus	X	0
A. taeniorhynchus	X	X
A. dorsalis	X	0
A. albopictus	0	0
A. nigromaculis	0	0

The prophylaxis of this disease is an important public health problem. As it has been shown that arthropods, principally mosquitoes, transmit the virus of the disease, it is important that measures be taken for their elimination. Control of mosquitoes and other insects should be carried out by proper screening of houses, barns, etc., by spraying and application of insecticides, adequate soil drainage, and destruction of breeding places. If the present concept is true, that the reservoir of the virus is in birds, elimination will present a difficult problem. Vaccination has been proved of value in animals and may be of value in preventing the disease in man.

There is no specific treatment. Serum from convalescent cases appears to be of little value, but this requires further study. General symptomatic treatment should be given.

What importance the disease will assume in the future in Louisiana is unknown. There seems to be no explanation of why the disease should remain dormant for years, then suddenly appear in new areas. Past experience, in epizootics among horses


SEASONAL DISTRIBUTION OF MOSQUITO VECTORS OF EQUINE ENCEPHALOMYELITIS, EASTERN TYPE
FOUND IN LOUISIANA - 1947
Legend:
NORTHERN & CENTRAL LA.
SOUTHERN LOUISIANA
JAN. A. aegypti DEC.
JAN. A. sollicitans DEC.
JAN. A. vexans DEC.
JAN. A. triseriatus DEC.
JAN. A. teaniorhynchus DEC.

and mules, has shown that rarely, except in a few endemic areas, does the disease recur to any extent in the same area in succeeding years. Human cases of equine encephalomyelitis usually occur only when an outbreak or epizootic of the disease is prevalent in the area, and usually after the peak in animals has been reached.

An infectious disease which has been responsible for only two proportionately small outbreaks, and a few sporadic cases in the past decade, might seem of little public health significance. However, it must be remembered that the distribution of the virus is widespread. If the present concept of the epidemiology of the disease is true, the possibility of infection in Louisiana is ever present.

SUMMARY

1). An outbreak of human equine encephalomyelitis, due to eastern type virus, occurred in Louisiana during 1947.

2). Ten cases were definitely proved to be of the eastern type, either by isolation of the virus from the brain, demonstration of neutralizing or complement fixing antibodies in the blood, by typical lesions in the brain, or by a combination of the above.

3). A study of the cases shows children were most frequently affected with no special sex predilection. The disease occurred in late summer and autumn, in small towns and rural districts, especially in the area where the disease was prevalent in animals.

4). Clinically the disease was severe, usually of short duration, with high case fatality rate. The gross and microscopic autopsy findings are given.

5). Etiologic diagnosis cannot be made on clinical findings. Diagnosis depends upon the laboratory and can be established by (1) Isolation of the virus from the blood, spinal fluid or brain tissue (2) test for neutralizing antibodies (3) complement fixation test and (4) microscopic examination of tissues.

6). Complete epidemiology of equine en-

cephalomyelitis, eastern type, is not definitely established. Evidence, at the present time, indicates that arthropods, particularly mosquitoes, transmit the disease, and birds act as the host reservoir.

7). Control measures include vaccination of animals against the disease, elimination of host reservoirs and insect vectors.

The author wishes to express appreciation to Dr. Emma Moss and Dr. M. Hood, of Charity Hospital, for assistance and cooperation in collection of data and specimens, and Dr. J. Arey, of Tulane University, for furnishing some of the tissue sections and lantern slides.

REFERENCES

1. Milner, K. C., Kelley, P. G., and Shaffer, M. F.: Human infection with virus of equine encephalomyelitis, eastern type, in Louisiana. N. O. M. & S. J., 100:270, 1947.

2. Feemster, R. F.: Outbreak of encephalomyelitis in man due to the eastern virus of equine encephalomyelitis. Am. J. Pub. Health, 28:1403, 1938.

3. Fothergill, L. D., Dingle, J. H., Farber, S., and Connerley, M. L.: Human encephalitis caused by the virus of the eastern variety of equine encephalomyelitis. New England J. Med., 219:411, 1938.

4. Webster, L. T., and Wright, F. R.: Recovery of eastern equine encephalomyelitis virus from the brain tissue of human cases of encephalitis in Massachusetts. Science, 88:305, 1938.

5. McAdams, J. C., and Porter, J. E.: Encephalitis in man caused by the virus of equine encephalomyelitis. New England J. Med., 221:163, 1939.

6. Hammon, W. McD.: Encephalitis: Eastern and western equine and St. Louis types, as observed in 1941 in Washington, Arizona, New Mexico and Texas. J. A. M. A., 131:560, 1943.

7. Bohls, S. W., and Irons, J. V.: Laboratory observation of virus encephalitis. Texas State J. Med., 39:260, 1942.

8. Sulkin, S. E.: Personal Communication.

9. Pollard, Morris: Personal Communication.

10. Piercy, P. L.: Personal Communication.

11. Getting, V. A.: Equine encephalomyelitis in Massachusetts. New England J. Med., 224:999, 1941.

12. Feemster, R. F.: Personal Communication.

13. Ten Broeck, C., and Merrill, M. H.: A serological difference between eastern and western equine encephalomyelitis virus. Proc. Soc. Exp. Biol. and Med., 31:217, 1933.

14. Merrill, M. H., Lacaillade, G. W., Jr., and Ten Broeck, C.: Mosquito transmission of equine encephalomyelitis. Science, 80:251, 1934.

15. Fothergill, L. D., and Dingle, J. H.: A fatal disease of pigeons caused by the virus of the eastern variety of equine encephalomyelitis. Science, 88:549, 1938.

16. Tyzzer, E. E., Sellards, A. E., and Bennett, B. L.: The occurrence in nature of equine encephalomyelitis in the ring-necked pheasant. Science, 88:505, 1938.

17. Ten Broeck, C.: Birds as possible carriers of the virus of equine encephalomyelitis. Arch. Path. 25:759, 1938.

18. Ten Broeck, C.: Transmission of equine encephalomyelitis. Third Internat. Cong. Microbiol. New York, 1939.

19. Davis, W. A.: A study of birds and mosquitoes as hosts for the virus of eastern equine encephalomyelitis. Am. J. Hyg., 32 (Sect. C):45-59, 1940.

20. Getting, V. A., and Feemster, R. F.: A survey of the mosquitoes of Massachusetts. Mass. Dept. of Public Health in Cooperation with the Work Projects Administration, 1940.

—o—

CHROMOMYCOSIS:

REPORT OF A CASE FROM LOUISIANA WITH A DISCUSSION OF ITS CLINICAL AND MYCOLOGIC FEATURES

LESLIE K. MUNDT, M. D.†

NEW ORLEANS, LA.

AND

MORRIS MOORE, PH. D.†

ST. LOUIS, MO.

In spite of several relatively recent articles on the subject, chromomycosis is still a disease with which many physicians are unfamiliar. Undoubtedly the condition is not as rare as it is generally supposed to be. More probably, the lesions have been misdiagnosed and confused with granulomas similar in appearance to one or more of its varied clinical forms. This report is submitted to call attention once again to chromomycosis, to review the clinical and mycologic features of the disease and to record the first such case from the state of Louisiana.

In 1911, in Brazil, Pedroso identified the first case of chromomycosis which he called "black blastomycosis" because of the dark fungous cells of the lesions.[1] Lane[2] and Medlar[3] described the first case in North America in 1915. Since that time approximately 100 cases have been recorded in the world literature, nine of which have come from the United States. The cases thus far reported have originated from widely scattered areas, including the Caribbean area (Cuba, Puerto Rico and Costa Rica), Brazil and the Union of South Africa.[4] In the United States, cases have been recorded from Massachusetts, Texas, Missouri, North Carolina, Pennsylvania and Georgia.[5]

Chromomycosis has been confused on many occasions with blastomycosis. Actual-

†From the Department of Dermatology and Syphilology, Louisiana State University Medical School, New Orleans, La., the Department of Dermatology, Touro Infirmary, New Orleans, La., and the Laboratory of Mycology of the Barnard Free Skin and Cancer Hospital, St. Louis, Mo.

ly, there are definite clinical and microscopic differential features between the two diseases, so that there can be little justification for a continuation of this common error in diagnosis.

Unlike blastomycosis, the chromomycosis organism divides, not by budding, but by cross wall or septum formation, both in culture and in tissue. To point out the difference between the two diseases and to avoid confusion in nomenclature, one of us (M. M.) with de Almeida has suggested the shorter term "chromomycosis" rather than "chromoblastomycosis" by which name it has been designated in the past, thereby omitting the misleading "blasto" altogther. The prefix in "chromoblastomycosis" and "chromomycosis" has been retained by all authors as a means of indicating the pigmented nature of the causative agent.

The organism in tissue has been described by Moore and his colleagues[6] as spherical or irregular sclerotic cells which are thick-walled, dark brown or chestnut colored and single or multiple, appearing in clusters. The cells produce by enlargement and septum formation to form mulberry-like groups. The cells are distributed throughout the tissue in microabscesses surrounded by polymorphonuclear leukocytes or engulfed by giant cells.

It is possible to make a clinical diagnosis only in far advanced typical examples of the disease. However, if the condition is kept in mind and biopsy resorted to, the diagnosis can be established histologically with no difficulty. The condition is undoubtedly more common than has been considered heretofore. More cases will be discovered in the future if physicians are aware of the world-wide distribution of the disease.

MYCOLOGY

The taxonomy of the fungi causing chromomycosis (chromoblastomycosis) has been somewhat confused by the succession of new names, genera, species and varieties. Morphologically, there can be seen various structures which may be found singly or in combination in all the organisms responsible for the disease. The one undisputed fungus, Phialophora verrucosa Medlar, 1915, is characterized by the production of semiendogenous spore formation through cupulliform structures termed phialophores. Other strains of fungi isolated from cases of chromomycosis have, in the past, been generally classified as Hormodendron pedrosoi Brumpt, 1921. The numerous isolated strains vary considerably morphologically, being characterized by the production of a variable number of large subspherical or ellipsoid, catenulate spores in acrogenous branches, the Hormodendron type of conidiophore; or the conidia may be arranged pleurogenously, along the conidiophore, or in head formation, the Acrotheca type of conidiophore; or there may be present the phialophores typical of Phialophora. In addition, one may find strains of organisms which have the formation of cupulliform spore bearers laterally or at the apices of the Hormodendron conidiophores.

Prior to the finding of the various methods of spore production and morphologic forms, the several isolated fungi were considered to be one organism and accordingly received the following names from different workers: Hormodendron pedrosoi Brumpt, 1921, Acrotheca pedrosoi Terra, Torres, da Fonseca and Arêa Leão, 1922, Trichosporium pedrosoi Langeron, 1929 and Gomphinaria pedrosoi Dodge, 1935.

In 1935, Carrión and Emmons[7] described semiendogenous spore formation in Hormodendron. In the same year, Moore and de Almeida[8] also reported all the methods of sporulation in one strain. On the basis of the various methods of spore formation and on the combination of these methods in various strains of fungi producing chromomycosis, Moore[9] in 1936 suggested a generic and genetic relationship of the fungi by placing the organisms in the order Phialophoreae and creating three new genera, Botrytoides, Hormodendroides and Phialoconidiophora. In the following year, Moore and de Almeida[10] established their relationship in accordance with the rules of botanical nomenclature.

In an antedated publication, Negroni[11] recognized the need for changing the name

Hormodendron. On the basis of a paper presented by Moore before members of the Bacteriological Institute, Buenos Aires, Argentina, in which the genus Hormodendroides was discussed, Negroni created the genus Fonsecaea to replace Hormodendroides which he considered a *nomen nudum*. The name honored da Fonseca, the eminent Brazilian mycologist who was one of the early workers on the disease. Carrión[12] accepted the name to replace the older terms and maintained two species, F. pedrosoi and F. compactum. Phialophora verrucosa was considered a distinct organism. In a later paper, Carrión[13] upheld the genus Fonsecaea and discarded all other genera. Following the phylogenetic sequence proposed by Moore, Carrión created varieties to replace the genera of Moore and de Almeida. F. pedrosoi var. communis replaced Phialoconidiophora guggenheimia, F. pedrosoi var. cladosporioides replaced Hormodendroides pedrosoi, F. pedrosoi var. typicus replaced Botrytoides monophora and F. pedrosoi var. phialophorica replaced Phialophora verrucosa. The term cladosporiodes is used by Carrión since it has been considered that Hormodendron is a synonym of the older Cladosporium.

Emmons, in presenting the mycologic report and discussion on an organism isolated from a patient with chromomycosis as published by Binford and Hess[14] rejected the genus Fonsecaea for the older name Phialophora. He preferred to amend the genus Phialophora to include, in addition to the semiendogenous spore formation, the acrogenous, pleurogenous or acropleurogenous spore formation described for Phialoconidiophora, Hormodendroides and Botrytoides. This seems to be a more logical choice than Fonsecaea. The new combinations, therefore, according to Emmons, in addition to the already accepted Phialophora verrucosa, would be P. pedrosoi and P. compactum. By amending Phialophora to include the other spore-forming types of organisms, Emmons recognized the close relationship existing between the various etiologic agents of chromomycosis as suggested by Moore, who placed all these fungi in the order Phialophoreae. Whether all the organisms should be placed in the genus Phialophora is a matter which may be open to discussion. Certainly the methods of sporulaion and the morphologic characteristics of Phialoconidiophora of Moore and de Almeida or Fonsecaea pedrosoi var. communis of Carrión would appear to merit more than synonomy with Phialophora pedrosoi as classified by Emmons.

The organism isolated from the case described here appears to have the characteristics of Botrytoides monophora or Fonsecaea pedrosoi var. typicus particularly when grown on Sabouraud's glucose agar and the characteristics of F. pedrosoi var. cladosporioides or perhaps Hormodendroides pedrosoi when grown on Czapek's medium. This would of course be classified as Phialophora pedrosoi by Emmons. On Sabouraud's glucose agar, pH 5.2, the fungus produces a colony which is compact with a central knob, concentric zones of growth, being dark olivaceous to black. The peripheral growth is dark greenish-gray to brown while the surface growth is dark mouse gray to olivaceous (Fig. 1:1). Growth is fairly rapid, attaining a diameter of approximately 3 cm. in 15 days. On Czapek's medium the colony is submerged in the agar, not compact, dark brown to black in color and attains a diameter of approximately 3 cm. in 15 days (Fig. 1:2).

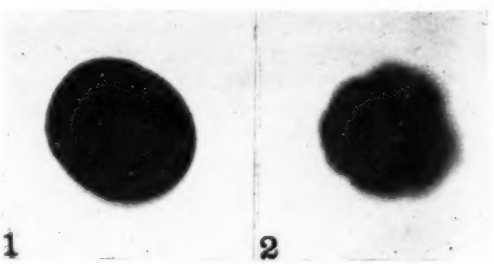


Fig. 1: 1. Colony of Phialophora pedrosoi on Sabouraud's glucose agar, 15 days old, actual size; (2). Colony of P. pedrosoi on Czapek's medium, 15 days old, actual size.

Microscopically, the fungus shows a paucity of the characteristic spore formation. On Czapek's medium the outstanding feature is the finding of brown spores, ellip-

soid to ovoid, catenulate, approximately 2-5 microns in diameter, developing from the hyphae laterally either singly or branched, suggesting the conidiophores of Cladosporium. Conidiophores of the Hormodendron

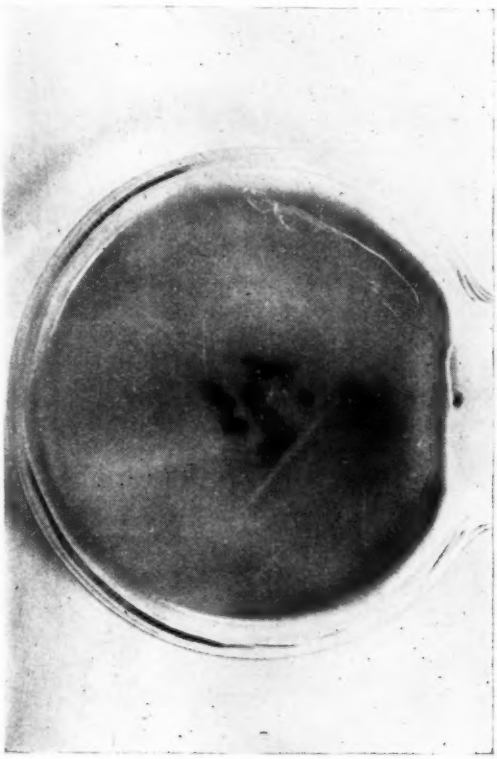


Fig. 2. Colonies of P. pedrosoi on Sabouraud's agar.

type may be seen, but not in great abundance and seldom attaining the development seen in other strains (Fig. 3:1-2). The conidia are subspherical to ellipsoid, catenulate in acrogenous branches, approximately 2-5 x 2-8 microns. Few conidiophores of the so-called Acrotheca type may also be seen. These are usually irregular, denticulate spore bearers, single or multicelled, lateral or terminal and dark brown with the conidia acrogenous or pleurogenous, ovoid ellipsoid or subfusiform, approximately 1.5-4 x 3-8 microns. On careful search, conidiophores of Phialophora may be found. These spore bearers, however, are rare. When observed in slide preparations they are usually simple, lateral on a hypha or occasionally branched showing two phialophores (Fig. 3:3-4). On Sabouraud's glucose agar the principal spore forming method is by the Acrotheca method (Fig. 3:5). The Hormodendron and the Phialophora types were not observed, although it is possible that they may be present.

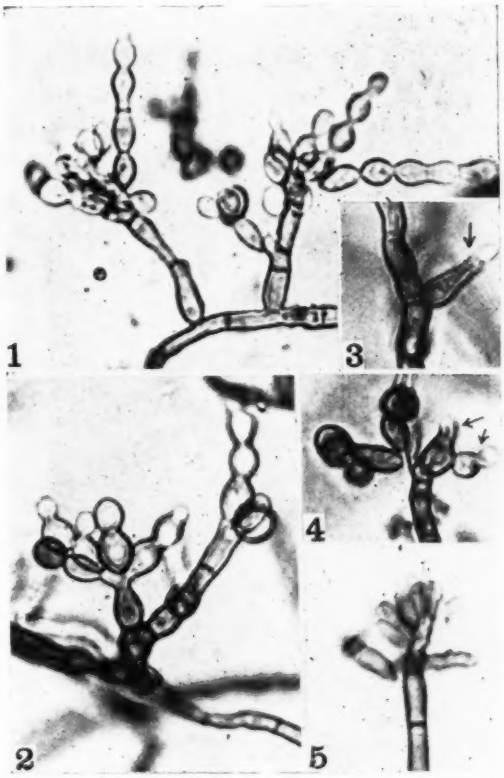


Fig. 3: 1. Hormodendron type of conidiophores grown on Czapek's medium, X 1440; (2). Conidiophore showing acrogenous spore formation on Czapek's medium, X 1440; (3). Semiendogenous type of conidiophore (Phialophora type) with single small conidium on Czapek's medium, X 1440; (4). Semiendogenous conidiophore (branching form) on Czapek's medium, X 1440; (5). Acrotheca type of conidiophore on Sabouraud's glucose agar, X 1440.

CLINICAL FEATURES

The clinical varieties of chromomycosis are manifold. Of the various species of the fungus considered causative, no one

species can be correlated clinically with any particular type of lesion.

Chromomycosis occurs in the laboring classes, mostly among farmers or those in contact with farm products. In many patients the history of injury by wood or vegetation can be elicited, as is probably the case reported in this paper.

The disease apparently may occur at any age. In the cases reported to date there has been a preponderant incidence in the male sex. It appears that all races are susceptible. The disease is not considered contagious since there has been no familial spread so far reported.

Fig. 4. Chromomycosis involving the index finger.

TYPES

In the past, chromomycosis has been considered a disease affecting the lower extremities in the form of verrucous and granulomatous excrescences. It is now known that any part of the cutaneous surface may be involved. Five clinical types have been described by Pardo-Costello and his associates.[15] These include:

1. The verrucous type, the form most frequently observed. This type is encountered usually on the legs in the form of papillomatous patches not unlike those seen in blastomycosis and tuberculosis verrucosa cutis. The color varies from light brown to dark red. The process tends to heal by central scarring. Suppuration is not a common feature.

2. The tuberculoid type also resembles tuberculosis verrucosa cutis as well as sarcoid. The typical lesion is a patch composed of brown hypertrophic nodules with erythematous areolas and minimal scarring. This type is considered to be an early form of verrucous chromomycosis. However, according to Pardo-Costello, the allergic response of the individual tissue to the infection may play an important role in the

Fig. 5. Same as Fig. 4. Enlarged view.

development of one or the other clinical types.

3. The syphiloid type resembles late cutaneous syphilis. The lesions are nodular, scaly and at times ulcerative, having a serpiginous, annular or arciform pattern. The process leaves a thin, depigmented scar.

4. The psoriasiform type appears as inflammatory infiltrated plaques with superimposed white, lamellated, adherent scales, similar in appearance to psoriasis.

5. The cicatricial and elephantiasic type is usually found on the leg and resembles Madura foot. This type is characterized by scarring and is considered a final stage of the disease, presenting, in addition to the scarring, verrucous lesions, scaling, ulcers and abscess formation. The elephantiasic enlargement is due to interference with the lymphatic drainage. Attacks of recurring lymphangitis are associated with this type.

COURSE

Chromomycosis is a chronic disease that progresses slowly, having been known to persist for as long as 40 years. There is no associated pain, pruritus or systemic symptoms in the majority of cases. Regional adenopathy is a common finding and is attributed to secondary infection. Metastases have been reported in only two cases and there have been no instances of invasion of bone. No visceral lesions have been observed. According to Pardo-Costello and and his associates,[15] atrophy of the muscles, ankylosis and osteoporosis are common in the extremities in cases of long duration, due to immobility and scarring. The general health of patients is unimpaired. A fatal case has never been reported except when caused by some unassociated condition.

PATHOLOGY

Microscopically, the diagnosis can be made by finding the typical cells in the tissue. There are many features microscopically which suggest blastomycosis, tuberculosis and the mycotic granulomas

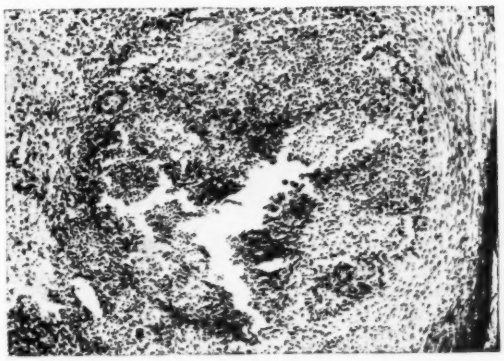


Fig. 6. Chromomycosis; suppuration and necrosis in a large abscess-like formation with fibrosis and a granulomatous response around the periphery. Low power. X 135.

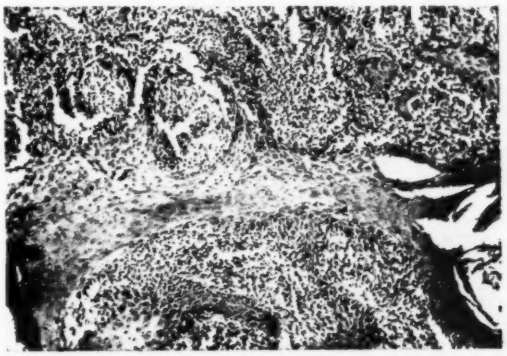


Fig. 7. Chromomycosis, showing pseudoepitheliomatous hyperplasia and microabscesses in the cutis. Low power. X 135.

Fig. 8. Chromomycosis, showing a group of the causative organisms. High dry. X 1280.

and there are certain characteristics which serve as differentiating points. The microscopic pathology has been described in detail by Moore et al as follows: "The epidermis shows hyperkeratosis and acanthosis with a thickening, broadening and elongation of the rete pegs. Within the pegs may be seen microscopic abscesses filled with polymorphonuclear leukocytes, cellular debris and the fungous cells. At times within these areas one may also find Langhans' giant cells containing fungi. The most pronounced changes are found in the dermis. Here one sees edema, a pronounced cellular infiltration and, in older lesions, evidence of fibrosis. The cellular infiltration is made up of polymorphonuclear leukocytes, lymphocytes, epithelioid cells, plasma cells, eosinophiles and Russell's fuchsin bodies, which have been noted in tissue sections by others. Giant cells of the foreign body type or Langhans' type may be present as well as macrophages. There is sometimes a tendency to pseudotubercle formation in the infiltrate. Fibroblastic changes may occur. Necrosis and microabscesses such as are found in Gilchrist's disease are not prominent in chromomycosis. The sclerotic cells of the fungus are found scattered throughout the corium, especially in the abscesses or in giant cells. They occur either as single cells or as mulberry-like clusters."

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

The diagnosis may be established by demonstrating microscopically, by the potassium hydroxide technic, the round, grouped, dark-colored sclerotic cells in pus, scales or sections of tissue. Cultural studies aid in reaching a diagnosis, but are not essential, since the typical cells can be demonstrated with ease and are pathognomonic of the disease.

The differential diagnosis includes blastomycosis, sarcoid, coccidioidal granuloma, maduromycosis, tuberculosis verrucosa cutis, bromoderma, syphilis, yaws, pyoderma vegetans, epithelioma, psoriasis and other diseases.

TREATMENT

Pardo-Costello et al.[15] say that roentgen therapy is of value in the treatment of the superficial type of case and is helpful even in other cases. They recommend from 600 to 1200 roentgen units filtered through 1 or 2 mm. of aluminum. According to these authors, iodides are worthless, as are thymol, antimony, copper and the sulfonamides. They state that the treatment of choice is electrocoagulation followed by curettage, which results in a firm contracted scar after two or three months of constant care. For the cicatricial and elephantiasic type on the legs only amputation is of any avail. In general, iodides in dosage up to 90 grains daily and roentgen therapy are usually tried, sometimes with success, as in the unpublished report of Sams. Moore, Cooper and Weiss[6] summarize the present thought on the subject by recommending iodides, roentgen therapy, electrocoagulation or surgical excision when the lesion is small and experimentative procedures when the lesion is extensive.

REPORT OF CASE

W. C., a white male, 81 years of age, who died on July 11, 1946, was followed at the Touro Infirmary, New Orleans, Louisiana, over a period of three years for carcinoma of the stomach and generalized carcinomatosis.

During the period from April 1945, until shortly before his death the patient was seen regularly in the hospital's dermatology out-patient department, service of Dr. R. A. Oriol, for a chronic dermatosis involving the proximal phalanx of the left index finger, of 15 months' duration. The dermatitis consisted of an erythematous plaque flush with the surrounding skin, having a slightly verrucous base without ulceration and covered with a loosely adherent white scale which tended to pile up at the border.

This condition resisted various types of local treatment as well as full doses of iodides and roentgen therapy. At the time of death, in spite of constant treatment, the dermatosis progressed slowly without at any time showing any tendency toward healing.

It was significant that the patient's occupation prior to the onset of the dermatitis consisted of working with raw wood from which barrels were constructed.

A biopsy made on September 26, 1945 was reported by Dr. J. R. Schenken as chromoblastomycosis. A description of this section of skin by Dr. Harvey Colvin was as follows: "Biopsy consists

of several small bits of skin presenting an intense chronic inflammatory and granulomatous reaction with scattered Langhans' giant cells. Pyogenic reaction is noted within the epidermis and subepidermis. Upon inspection with the high power lens, several rounded brown forms may be seeen morphologically consistent with chromoblastomycetes. These sometimes occur singly but one group of four is noted together. These forms are most numerous along the edges of small pyogenic abscesses within the corium."

Scrapings from the involved area yielded the causative fungus of chromomycosis described above.

SUMMARY

A case of chromomycosis, thought to be the first from Louisiana, is reported. The disease is discussed from the standpoint of both its clinical and mycologic features. In spite of the paucity of reports, the disease is probably not rare. It is suggested that physicians be on the lookout for further cases from this section of the country.

REFERENCES

1. Weidman, F. D., and Rosenthal, L. H.: Chromoblastomycosis: A new and important blastomycosis in North America, Arch. Dermat. & Syph., 43:62, 1941.

2. Lane, C. G.: A cutaneous disease caused by a new fungus (Phialophora verrucosa), J. Cutan. Dis., 33:840, 1915.

3. Mediar, E. M.: A cutaneous infection caused by a new fungus, Phialophora verrucosa, with a study of the fungus, J. Med. Research, 32:507, 1915.

4. Snow, J. S., Wedding, E. S., and Tomlinson, W. J.: Chromoblastomycosis, Arch. Dermat. & Syph., 51:90, 1945.

5. Emmons, C. W., Hailey, Howard, and Hailey, Hugh: Chromoblastomycosis, J. A. M. A., 116:25, 1941.

6. Moore, M., Cooper, Z. K., and Weiss, R. S.: Chromomycosis, J. A. M. A., 122: 1237, 1943.

7. Carrión, A. L., and Emmons, C. W.: A spore form common to three etiologic agents of chromoblastomycosis, Puerto Rico J. Pub. Health & Trop. Med., 11:114, 1935.

8. Moore, M., and Almeida, F. de: Etiologic agents of chromomycosis (chromoblastomycosis of Terra, Torres, Fonseca and Leão, 1922) of North and South America, Rev. de Biol. e Hig., 6:94, 1935.

9. Moore, M.: Chromomycosis, Folia Bil. (Buenos Aires, Argentina) Nos. 61-65:266-269, 1936.

10. Moore, M., and Almeida, F. P. de: New organisms of chromomycosis, Ann. Missouri Bot. Gard., 23:543, 1936.

11. Negroni, P. :Estudio micológico del primer caso argentino de cromomicosis Fonsecaea (n. g.) pedrosoi (Brumpt, 1921), Rev. Inst. Bact. del Depto. Nacional de Hig. (Buenos Aires), 7:419, 1936.

12. Carrión, A. L.: The specific fungi of chromoblastomycosis, Puerto Rico J. Pub. Health & Trop. Med., 15:340, 1940.

13. Carrión, A. L.: Chromoblastomycosis, Mycologia, 34: 424, 1942.

14. Binford, C. H., Hess, G., and Emmons, C. W.: Chromoblastomycosis. Report of a case from continental United States and discussion of the classification of the causative fungus, Arch. Dermat. & Syph., 49:398, 1944.

15. Pardo-Costello, V., Leon, E. Rio, and Trespalacios, F.: Chromoblastomycosis in Cuba, Arch. Dermat. & Syph., 45:19, 1942.

TRACHEOTOMY WITH COMPLICATION OF PNEUMOTHORAX

ASHTON THOMAS, M. D.†
NED W. HOLLAND, M. D.†

NEW ORLEANS

Tracheotomy, even when performed as an emergency measure, is in most instances free of complications, but when they do occur trying moments are experienced by the physician. The two cases to be presented aptly demonstrate the serious complications which may ensue,—in this instance, pneumothorax, which followed tracheotomy on two children, one of whom survived, and the other expired.

Case No. 1. A seventeen months old white male was admitted to Charity Hospital with the chief complaint of cyanosis, cough, and fever. His present illness dated back three days, at which time cough and fever were first noted. The cough had become more severe with each paroxysm, with associated inability to get his breath. The day before admission, cyanosis following such a paroxysm had been noted. The cough and croup had become more pronounced, together with a loud inspiratory noise, which led the child to be admitted for treatment.

Physical examination revealed in general a toxic appearing child with a loud inspiratory croup, a spasmodic cough, and moderate intercostal and substernal retraction. The temperature was 101° F. per rectum. The pharynx was injected, with no evidence of membrane formation. The remainder of the physical examination was essentially negative. The diagnosis made at this time was acute laryngo-tracheobronchitis.

The child was placed in a croup tent, receiving steam inhalations, oxygen, and sedation of phenobarbital, supplemented with penicillin intramuscularly, and sulfadiazine orally. Six hours later no improvement was noticed, instead the respirations were more labored. Tracheotomy was deemed advisable, and over a bronchoscope, a low tracheotomy was performed. Bronchoscopy revealed a hyperemic larynx and trachea with a moderate amount of seromucinous exudate present in the tracheobronchial tree. Immediate postoperative relief was obtained. Twenty-four hours later, breath sounds over the left chest were found to be diminished along with an increase in respiratory rate. X-ray of the chest revealed a left pneumothorax and emphysema of the left lung. A water trap

---

Presented before the Otolaryngological and Ophthalmological Society at Charity Hospital of Louisiana.

†From the Charity Hospital of Louisiana and the Louisiana State University School of Medicine.

tube was inserted into the left pleura with removal of 175 cu. c.c. of air, and moderate relief of symptoms. Within twelve hours this was discontinued as the child was greatly improved. Eight days later, after an apparently normal recovery, with the tracheotomy tube being blocked eighteen hours, the tube was removed, but shortly afterwards the child began to experience severe dyspnea. An attempt was made to reinsert the same tracheotomy tube, but was unsuccessful. Therefore, again over a bronchoscope, opening the original wound, the tube was reinserted. Within twelve hours a pneumothorax again was detected on the left side after the same treatment as previously mentioned. After twelve days of uneventful recovery, the tracheotomy tube was finally removed, and the patient was discharged shortly afterwards.

Case No. 2. A white female, 2½ years of age, was admitted to Charity Hospital with a chief complaint of difficulty in breathing. The present illness dated back three days, with hoarseness the only complaint. The following day slight difficulty in breathing, and fever, had been noted, but no cough. This complaint continued to get worse until ten hours prior to admission when cough also was present.

Physical examination at the time of admission revealed in general an acutely ill child with marked respiratory difficulty, substernal and suprasternal retraction with each inspiratory effort, and cyanosis about the hands and mouth. Her temperature was 103° F. The pharynx was hyperemic, with evidence of a gray membrane on the left palatine tonsil. Otherwise, the physical examination was essentially negative. A tentative diagnosis of diphtheria was made.

Tracheotomy was deemed advisable, and over a bronchoscope, a low tracheotomy was performed, the child experiencing marked relief immediately afterwards. Bronchoscopy revealed hyperemia of the larynx with no membrane formation. One hour and a half later the child exhibited signs of air hunger, cyanosis, rapid pulse, and increased respiratory rate. Subcutaneous emphysema of the neck anteriorly and laterally was also palpable. Rebronchoscopy through the tracheotomy opening revealed no obstruction. X-ray, however, did show a massive bilateral pneumothorax and mediastinal emphysema. Both pleura were immediately tapped and 420 cu. c.c. of air withdrawn, following which 240 c.c. of air were withdrawn from a similar tap into the mediastinum. Water traps were connected bliaterally, but even with these measures, the patient expired four hours later. At autopsy a diagnosis was made of exudative tracheobronchitis, bilateral pneumothorax with mediastinal emphysema, and bronchopneumonia.

Tracheotomy is followed by complications much more frequently than a review of the literature shows. If there is no violation of technic in performing a tracheotomy, complications should be rare. The cases presented here, of mediastinal emphysema and pneumothorax, represent a complication which occurs more often than one is apt to believe. Neffson reports that 13.5 per cent of a series of cases with nondiphtheritic obstructive infections developed pneumothorax.

There are several possible causes for the development of these complications, such as direct injury to the pleura, or rupture of alveoli into interstitial pulmonary tissue with migration along the peribronchial and perivascular spaces to the mediastinum and eventual rupture into the pleural space. The most likely cause is excessive inspiratory movements with sucking of air into the mediastinum and eventual rupture into both pleural spaces. The usual explanation is that air enters the mediastinum through the wound in the cervical fascia, becomes imprisoned in the mediastinum, (especially when obstruction is not relieved), and finally reaches a tension high enough to rupture the mediastinal pleura, which is especially thin in children. When there is pneumothorax there always seems to be a coexisting mediastinal emphysema. Pneumothorax is usually bilateral. Jackson believes mediastinal emphysema is usually due to tight closure of the wound around the canula. Spontaneous pneumothorax has occurred from rupture of the visceral pleura in violent respiratory effort during impending asphyxia and is more likely to occur when previous adhesions exist.

The most frequent cause seems to be sucking of air through the tracheotomy wound into the mediastinum. Therefore, prevention of this is important, and can be accomplished by the observance of certain precautions, one of which is to eliminate the possibility of respiratory obstruction during operation. Avoid giving general anesthesia until the bronchoscope is in place, otherwise asphyxia may ensue inasmuch as the accessory respiratory muscles are put at rest and there is no voluntary stimulus. A bronchoscope in place before converts a gasping, restless, struggling child into a

quiet one, and the operation can proceed unhurried. It is well also to desist from opiates, too small an incision, or closing too tightly. Aspirate pathways frequently; however, postoperative obstructions may occur despite heroic measures. Another important precaution is to avoid the opening of avenues for air to get down. Anatomic dissection with demonstration and spreading wide of each distinct fascial layer, particularly pretracheal, should be avoided. Pretracheal fascia and cartilaginous rings should be cut at the same time and from below upward. Application of iodoform packs about the wound to seal off the spaces is considered very effective.

The time element, that is, when to perform the tracheotomy, has an important bearing on the success of the procedure. Michels believes that operation should be done as soon as adequate indications arise, not waiting for extreme conditions. Early diagnosis is essential inasmuch as time means so much, and is determined by the clinical picture, the physical signs, and the use of x-ray. The clinical picture may be so slight as to be unrecognized, with the patient rather free of symptoms, and yet, on the other hand, the patient may develop shock, cyanosis, and asphyxia in short order. The physical signs are generally severe dyspnea in which there is diminution in the volume and the force of the air through the tube. There is diminished excursion of the affected side with displacement of the heart to the opposite side. There is diminution of breath and voice sounds with hyperresonance and tympany. X-ray should be done as soon after tracheotomy as possible.

TREATMENT

When the symptoms are mild, careful watch should be exercised as air may be absorbed. When there is tension pneumothorax (air can get in but cannot get out) repeated decompression is necessary. Failure gives anoxia from massive venous stasis due to increased intrathoracic pressure. Decompression should be done gradually without too great a shift in mediastinal pressure, or air embolism may occur. Decompression should be done with a special apparatus, or even with the pneumothorax machine and oxygen over the tube. Jackson suggests prompt bronchoscopic oxygen insufflation, constantly maintained until at least one lung has reexpanded.

Closed drainage of the chest may be necessary. Most observers feel that mediastinal surgery has met with only fair success and is to be avoided.

In unilateral pneumothorax, the mortality is usually less than 20 per cent as contrasted with bilateral pneumothorax in which the mortality is seldom less than 90 per cent.

Other complications, such as pneumonia and wound infections are usually treated with the sulfa drugs and penicillin. Frequently there are situations which arise and which appear to be pneumonias. These are actually areas of pneumonitis associated with atalectasis, in which the latter is due to plugs of mucus and similar concretions. Removal by the bronchoscope relieves this condition. Laryngeal and tracheal stenoses have been recently discussed and are usually due to high tracheotomy or misfit canula. As a means of preventing these it is well to cut below the second ring of trachea. The stenoses are associated with destruction of cartilaginous framework and may be corrected with core molds.

REFERENCES

1. Boyson, G. and F.: Acta Oto-Laryngology, 33:50-54, 1944.

2. Johnson, L. F.: Ann. Otol., Rhinol., and Laryngol., 53:837-838, 1944.

3. Whigham, J. R. M.: Brit. Med. Jour., 2:47, 1945.

4. Neffson, A. H.: Arch. Otol., 36:773-815, 1942.

5. Forbes and Salmon: Jour. Pediat., 23:175-183, 1943.

6. Work, M. P.: Arch. Otol., 37:526-535, 1943.

7. Neffson, A. H.: Arch. Otol., 37:23-29, 1943.

8. Jaffe, L.: Arch. Otol., 34-832-834, 1941.

9. Goidberg and Mitchell: Am. Jour. Surg., 56:448-454, 1942.

10. Jackson and Jackson: Diseases of Ear, Nose, & Throat, 1946.

MYOCARDIAL ANEURYSM CAUSING ESOPHAGEAL OBSTRUCTION

REPORT OF CASE PRESENTING A DOUBLE CARDIAC ANEURYSM WITH SEVERE DYSPHAGIA NECESSITATING GASTROSTOMY

MORRIS ISAACSON, M. D.*

JACKSON, MISS.

Myocardial aneurysm involving the posterior wall of the left ventricle is not an uncommon finding secondary to myocardial infarction. Autopsy studies show involvement of this region in approximately 10 to 20 per cent of the cases in which ventricular aneurysm occurs.[1, 2] In about 10 per cent of all cases more than one eneurysm is found. Dysphagia, when present, is rarely a prominent symptom. The following case of double cardiac aneurysm with dysphagia and subsequent malnutrition severe enough to require gastrostomy is, therefore, considered worthy of note. A search through the literature fails to disclose a similar case. However, Strandell[3] describes an interesting case of aneurysm of the apex of the left ventricle, diagnosed, in vitam, in a luetic musician, aged 42. This patient complained of dysphagia characterized by a stinging pain in the lower chest four seconds after swallowing a mouthful of food. At autopsy, the discovery of a second aneurysm of the posterior wall of the left ventricle explained the patient's dysphagia for which there had earlier been no adequate explanation.

CASE REPORT

H. M., a 52-year-old, ambulant, colored male, veteran of World War I, was admitted to the Veterans Administration Hospital, Alexandria, La., on October 22, 1946, as a transfer from the U. S. Marine Hospital, Galveston, Texas, with symptoms of choking sensation in the suprasternal region, vomiting and soreness at the site of attachment of his gastrostomy tube to the abdominal wall.

He had no illness of consequence in childhood. His family history was non-contributory. Prior to his Army service he worked in several sawmills. He was married three times. In 1911, he contracted gonorrhea, which he treated himself with oral medication, and the following year he had a penile "haircut" which healed in a few days with applications of "bluestone" water. While in the Army he was found to have a positive blood Wasserman reaction for which he was given about thirty injections. In August 1918, he was hospitalized two weeks for complaints of headache, fever, malaise and generalized joint aching, and was diagnosed as having "tertiary" syphilis.

Following his discharge from the Army he worked in several logging camps as a "tong hooker" until 1939, then was employed as team driver, carpenter's helper and common laborer in an oil refinery, in shipyards and in lumber mills until December 1944. A blood Wasserman test in 1924 was negative. He drank "right smart" of whiskey, averaging about a quart weekly, from 1920 to 1935, but in the latter year he "quit drinking." His health prior to 1928 was fairly good except for occasional "rheumatism" pains in his knees and shoulders and "biliousness" which was usually relieved by purgatives.

In the early part of 1928, while he was engaged in loading logs, his tong slipped from the end of a log which was being hoisted, and he was jerked forwards. A few minutes later, he experienced a sharp, intermittent, non-radiating, tingling pain just below the cardiac apex. This pain, unlike anything he had ever experienced before, lasted for about ten to fifteen minutes, then gradually subsided. Despite the pain, however, he continued his work. He had no further trouble until two weeks later when the pain recurred, again lasting for several minutes. From then on, for the next six months, the pain recurred at shorter and shorter intervals, and its duration progessively increased until it would occur every few days and last for three to four hours at a time. Associated with this pain he noticed increasing dyspnea on exertion. These symptoms finally obliged him to give up his work and to enter the Charity Hospital in New Orleans. After a week's treatment with potassium iodide and corrosive sublimate, he began to improve symptomatically, and, in three weeks he was well enough to leave the hospital. Two weeks later he returned to his former occupation.

From 1928 to 1935, he experienced infrequent lower abdominal cramping pains after meals; these pains were usually relieved by soda, vinegar, or laxative tablets. In the early summer of 1935, while he was seated on the commode one afternoon, he felt "hot and faintified" and perspired profusely. He called for help and slumped backwards but did not lose consciousness. Following this incident he felt dizzy for an hour and stayed in bed for a few days. He then noticed increasing dyspnea on walking and working. In addition, his cramp-

*From the Medical Service of the Veterans Administration Hospital, Alexandria, La.

Published with permission of the Chief Medical Director, Department of Medicine and Surgery, Veterans Administration, who assumes no responsibility for the opinions expressed or conclusions drawn by the author.

ing abdominal pains recurred more frequently, on the average of once every two or three months, and would last a day or two before subsiding. He decided at this time to quit drinking alcohol. Nothing further of note occurred until the latter part of 1940, as he was talking to a mill owner in a lumber office, he suddenly lost consciousness and fell to the floor. When he recovered from this syncopal attack several minutes later, he felt dizzy and weak and had a mild headache. Following this episode, which was not attended either by paralysis or convulsions, he stayed in bed for a few weeks.

In the spring of 1942, following a routine blood serological test at a Health Clinic, he was told that he needed "shots" for his blood. He was given weekly intravenous and intramuscular injections for a period of five months and, at the end of this time, had a negative Wassermann. In July 1944, his cramping lower abdominal pains became so severe that he visited several physicians for relief. X-ray studies were made, revealing an ulcerated stomach, and he was admitted to the City Hospital in San Francisco for treatment. Under Sippy regime, his pains gradually subsided and, after about a week, he was discharged from the hospital. Within a few days, he returned to his work. On September 27, 1944, as he was stacking lumber, he suddenly felt excruciating pain to the right of his umbilicus and had a sensation of extreme weakness. He was readmitted to the City Hospital, where he was immediately operated upon and found to have a perforated ulcer. His postoperative course was uneventful; the lower abdominal pains, which had been bothering him since 1935, disappeared, and, after fifteen or sixteen days, he felt well enough to leave the hospital. Six weeks later, he returned to work in a sawmill. He worked until December 21, 1944, when he slipped while climbing a stack of lumber and injured his back.

In July 1945, he had almost completely recovered from his injury when he experienced a siege of hiccoughs and vomiting which lasted continuously for a week. For a few days prior to this episode, he felt "droopy and weak," and "couldn't stand to walk much." One evening, towards 11:00 P. M., he awoke feeling "sick in the stomach," then commenced to vomit and to hiccough. His vomitus consisted at first of food, then of a slimy yellowish liquid. He was unable to retain even liquids. After a week's vomiting and hiccoughing, he had a respite for a day or two; then the symptoms recurred. There was very little nausea associated with his vomiting. He began to lose considerable weight as a result of his vomiting, and sought hospitalization.

During September, and again during October, 1945, he was treated at the U. S. Marine Hospital, Galveston for duodenal ulcer, with little relief of his symptoms. He was readmitted on December 17, 1945, when an electrocardiogram was made, showing evidence of an old posterior wall infarction. Diagnoses of duodenal ulcer and aneurysm of the left ventricle due to infarction were then made. Under treatment with Sippy and liquid regime, the patient showed some symptomatic improvement but was never entirely free of his choking sensation or vomiting. Ten days after his discharge from the hospital on March 2, 1946, he had to be readmitted. His weight by this time had dropped to 114 pounds as compared with his normal weight of 165 pounds at the onset of his illness in July 1945. Gastric analysis showed a free HCl of 118 degrees. Fluoroscopic study and a gastro-intestinal series disclosed a practically complete esophageal obstruction behind the heart shadow, with barium unable to pass beyond this point. A calcified shadow was noted in the region of the left ventricle. The roentgenologist's impression was carcinoma of the esophagus. In view of the patient's marked emaciation, a gastrostomy tube was inserted for feeding purposes on April 12. The patient was now able to retain part of his nourishment. Barium study of the esophagus was repeated on June 27; it now revealed an incomplete obstruction. There was a persistent, smooth, symmetrical constriction of the esophagus "posterior to the portion of the cardiac silhouette formed by the right auricle," with delay of barium at this point. The previous impression of stricture on a carcinoma basis was now considered doubtful.

In July 1946, the patient was given a week's furlough during which time he adhered to a soft diet but took no gastrostomy feedings. Although he still had some vomiting while at home, his general condition remained essentially unchanged. Upon his return to the hospital, it was felt that his tube could be removed, and, accordingly, on the 25th of July the gastrostomy tube was removed and the opening closed. The patient appeared to be getting along fairly well when, on August 1st, after eating a few mouthfuls of roast meat, he had recurrence of his vomiting. On August 5th, his present gastrostomy tube was inserted. On August 15th, esophagoscopy was performed, and a portion of tissue was removed for biopsy. No tissue suggestive of carcinoma or other malignancy was found. At the esophageal-gastric junction, the mucosa appeared granular. A diagnosis of cardiospasm was then made. A repeat fluoroscopic study on August 22nd disclosed that the calcified mass overlying the cardiac silhouette in the lower portion of the left chest was not attached to the diaphragm and showed no pulsations, either intrinsic or transmitted. The roentgenological impression was benign mass, lying either within the pericardial fat or in the portion of the lung anterior to the pericardium.

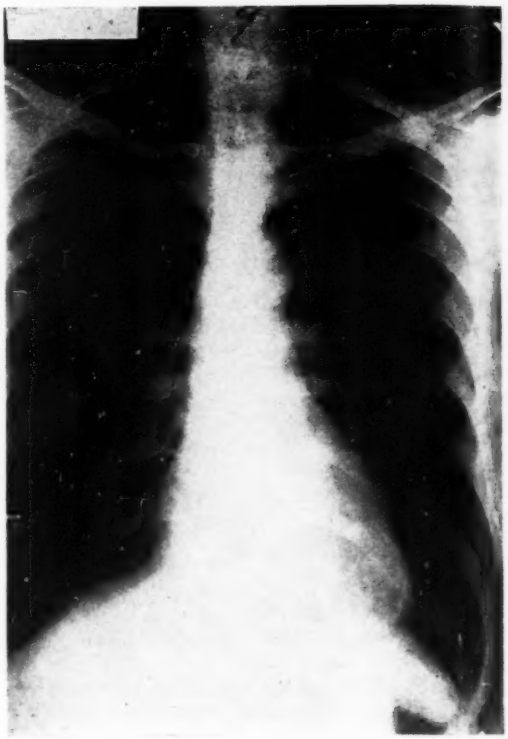
In the latter part of August, patient was sitting in a wheel chair when he suddenly began to feel

"dizzy and hot," and "passed out." When he regained consciousness several minutes later, he felt weak and dizzy; these symptoms persisted for several hours. Injections of novocaine into the dorsal sympathetic chain were considered for relief of his cardiospasm, but he repeatedly refused to have these performed because of his weakness. He was then transferred to the Veterans Administration Hospital in Alexandria, La., for domiciliary care.

Examination on admission revealed a chronically ill-appearing, ambulant colored male, height 6′ 2″, weight 125 pounds, who had an indwelling gastrostomy tube. His chief complaints were occasional choking sensation in the suprasternal region, vomiting, and soreness at the site of insertion of his gastrostomy tube. His vomiting bore no fixed relation to his meals; it would occur at any time of day or night. He described its onset as follows: "A choking comes on me (pointing to suprasternal notch) and cuts my wind off. Seems like if I can belch, it gives me all the relief. Otherwise, I has to vomit." His vomiting rarely exceeded two mouthfuls of a slimy fluid, which was occasionally mixed with food. There was little nausea, and no burning or bitter sensation, associated with his vomiting. His vomitus never showed any blood. After a vomiting spell, he would stay as quiet as possible for two or three hours, at the end of which time he would generally "catch his breath" better.

He appeared somewhat pallid, but was not dyspneic at rest. His thorax was long and slightly emaciated. The point of maximum impulse was visible and palpable in the 6th intercostal space, ½ cm. lateral to the left nipple. Upon palpation, a definite expansile impulse was felt at the apex. Contrasting with this finding, the first heart sound was faint over all cardiac areas. The second apical sound was clicking in character. A_2 was louder than P_2. The heart rhythm was regular, except for the presence of occasional extrasystoles. No thrill was palpable. A soft, blowing, non-radiating, late systolic murmur was heard at the apex and at the aortic area. Upon moderate pressure with the bell-type stethoscope in the region of the apex, with the patient in the upright position, a short scratching sound was heard during late systole. This sound was inconstant, however, and disappeared when patient was in the supine position. The pulses were equal and synchronous. There was no undue peripheral arteriosclerosis. The pulse rate was 78 per minute. Blood pressure readings in both arms were 114/84. The lungs were negative. Other physical findings were essentially negative.

X-ray of the chest in the PA view (Fig. 1) revealed a cardio-thoracic ratio of 53 per cent, and an ovoid area with calcified border measuring approximately 5.5 cm. by 4.5 cm. near the apex of the heart. On fluoroscopic examination, an expansile pulsation was visible on the posterior



Figue 1. PA view of chest showing aneurysm with intramural calcification at the apex of the left ventricle.

aspect of the heart. The esophagus was found to be constricted in its distal third; this constriction was apparently due to pressure from an aneurysm of the posterior portion of the heart. Above the point of constriction, the esophagus appeared slightly dilated (Fig. 2). No defects, tenderness, or niche formation were noted in the stomach or duodenal cap. The stomach was entirely empty at the end of one hour. The gastrostomy tube was located in the distal third of the lesser curvature. An electrocardiogram made on admission showed evidence of an old posterior wall infarction, with coronary-type inversion of T_2 and T_3, a Q_2T_2 and Q_3T_3 pattern, and occasional premature ventricular contractions (Fig. 3). The blood Wassermann and Kahn were negative. The blood count on admission showed a hypochromic anemia, with an erythrocyte count of 3,860,000 and a hemoglobin of 54 per cent.

Patient was given a transfusion of 250 cc. citrated blood on November 4th. He was placed on a soft diet and fed, in addition, per gastrostomy tube, 150 cc. tid of a 1000 calorie formula consisting of milk, sugar, tomato juice, whole eggs and brewer's yeast. Medication consisted of tincture of bella-

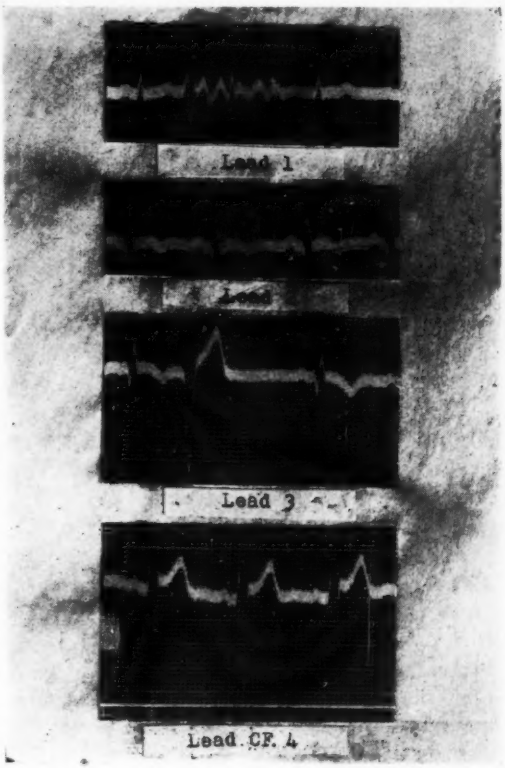
Figure 2. Left anterior oblique view showing dilatation of esophagus above area of construction resulting from aneurysm of the posterior wall of the left ventricle. Note intramural calcification at apex of left ventricle.

donna, 10 drops tid, and nitroglycerin tablets, gr. 1/100, sublingually, prn.

Under this treatment, he showed definite symptomatic improvement. His weight increased to 137 pounds. Gastric analysis of a fasting specimen showed a free HCl of 40 degrees and a total acidity of 78 degrees. His appetite was good. Although there was a marked lessening of the discomfort resulting from his choking sensation, he still experienced, once or twice a week, some choking on swallowing; this was relieved by his vomiting. The nitroglycerin sublingually appeared to be of definite value in diminishing his choking sensation. Early in December his tube feedings were discontinued for a period of two weeks. During this time his weight remained stationary. In the latter part of December, he had another syncopal episode; while sitting near his bedside he suddenly felt "weak, dizzified, and blind", then the next thing he remembered was finding himself in bed. At the Christmas holidays he was given a two weeks' furlough, during which time he supplemented his oral feedings with sweetened milk through his gastrostomy tube. Upon his return to the hospital, his general condition was satisfactory. At the present time, he is freely ambulant, but experiences slight dyspnea when he walks from his ward to the gate, a distance of approximately half a mile.

COMMENT

This case presents several unusual features. The patient's history and his symptoms of recurrent precordial pain, increasing in intensity and duration over a period of several months, strongly suggests that he had an initial coronary occlusion in 1928. Despite his pain, he continued his arduous work. The added strain thus imposed upon a weakened myocardium, it is reasonable to suppose, resulted in the formation of an aneurysm at the apex of the left ventricle, the most frequent site for such a lesion. Fulton[4] has called attention to the significance of recurrent, prolonged pain follow-



Figue 3. Electrocardiogram taken 10-23-46 showing evidence of old posterior infarction and occasional premature contractions, one of which is interpolated.

ing the initial episode in the diagnosis of cardiac aneurysm, and has stressed the importance of insufficient rest following myocardial infarction in its causation. Evidence of this initial aneurysm is now furnished by the ovoid mass with intramural calcification at the apex. The absence of pulsation is explained by the intramural thrombus formation and calcium deposit.

A second aneurysm then occurred, probably in July 1945, this time involving the posterior wall of the left ventricle and causing a marked constriction of the lower third of the esophagus. Considering the more or less sudden onset of this patient's dysphagia, the evolution of this aneurysm must have been rapid. Evidence of this second aneurysm is obtained from fluoroscopic study, which reveals an expansile pulsation on the posterior aspect of the heart, and from the electrocardiogram, which shows evidence of an old posterior wall infarction.

Since esophageal obstruction due to aneurysm of the posterior wall of the left ventricle is such a rare entity, it is not surprising that carcinoma of the esophagus was at first suspected as the etiological factor. Further studies, however, cast doubt upon this diagnosis. A diagnosis of achalasia was then made. The significance of the calcified mass in the apical region was overlooked. It was thought to be a benign tumor, lying either within the pericardial fat or in the lung tissue anterior to the pericardium. Some significance should be attached to this patient's syncopal episodes, the first two occurring before, and the second two subsequent to, his perforated ulcer. Among the causative factors to be considered are minute cerebral emboli arising from an aneurysmal mural thrombus, cerebral anemia secondary to paroxysmal cardiac arrhythmia, cerebral arteriosclerosis and, possibly, luetic cerebral endarteritis.

In the association of syphilis with double cardiac aneurysm this case bears a striking resemblance to the case described by Strandell. Another feature of interest is the patient's state of relative well-being at the present time. If, as appears likely, his initial myocardial infarction and aneurysm occurred in 1928, then he was able, nevertheless, to engage subsequently in arduous activities for a period of sixteen years. Although mild esophageal obstruction has been noted occasionally in cases of aneurysm of the posterior wall of the left ventricle[3, 5], it is very unusual for the dysphagia to be so prominent a symptom as to require gastrostomy.

SUMMARY

1. An unusual case of double cardiac aneurysm in a 52-year-old colored male is reported, with a predominating symptom of dysphagia of such severity as to require gastrostomy for feeding purposes. The first aneurysm probably occurred in 1928, as a sequela to coronary occlusion, and is now recognizable by a calcified, ovoid, nonpulsating mass at the apex of the left ventricle, the most common site for cardiac aneurysm. A second aneurysm then developed, probably in July 1945, this time involving the posterior wall of the left ventricle, and causing a marked constriction of the lower third of the esophagus. This aneurysm is demonstrable by fluoroscopy and its etiology is confirmed by the electrocardiogram, which reveals evidence of an old posterior wall infarction.

2. Relative well-being and an active life are not incompatible with aneurysm of the heart. In the present case, the patient engaged in the arduous labor of a logger and team driver for a period of sixteen years following the occurrence of his initial aneurysm.

3. Pressure upon the esophagus from a cardiac aneurysm, although rare, should be considered in the differential diagnosis of dysphagia. In the present instance, the patient's symptoms were at first attributed to carcinoma of the esophagus, then to achalasia.

The author is indebted to Dr. Oza J. LaBarge, Chief of the Medical Service, and to Drs. Arthur G. Sullivan, Allen C. Winters, and Samuel A. Shelburne for their interest and helpful criticisms.

REFERENCES

1. Appelbaum, E. and Nicolson, G. H. B.: Occlusive disease of the coronary arteries, Am. Heart J., 10:662, 1935.
2. Schwedel, John B.: Clinical Roentgenology of the

Heart. Ann. of Roentg., XVIII: 87, 1946 (Paul B. Hoeber, Inc.).

3. Strandell, Birger: A contribution to the diagnosis of aneurysm of the heart. Acta Med. Scand., 74 (2) :165, 1930.

4. Fulton, M. N.: Aneurysm of the ventricle of the heart. J. A. M. A., 116:115, 1941.

5. Wolferth, C. C. and Wood, F. C.: Acute cardiac infarction involving anterior and posterior surfaces of left ventricle. Arch. Int. Med., 56:80, 1935.

PAPILLOMA OF THE GALLBLADDER: CASE REPORT

GORDON McHARDY, M. D.
AND
EDWIN EDWARDS, M. D.
NEW ORLEANS

The relative rarity of cholecystic papilloma has been previously presented by one of us.[1] Kirklin's[2] diagnostic roentgen accomplishments are recognized. A single case study[3] since ours likewise was diagnosed roentgenologically and comments were made of the possible, but unlikely, relationship to carcinoma of the gallbladder. We take issue with Greenwald's disregard for malignant potentiality on the basis of prevalence of calculosis with carcinoma of the gallbladder, while rarely are papillomas associated with calculi. Generally, while the frequent association is recognized it is conceded that the presence of calculi is merely incidental to the diagnosis of cholecystic malignancy; one could not on this basis discount malignant potentiality of a gallbladder papilloma. Phillips[4] reports calculi complicating his reported cases of papilloma.

An important point emphasized by Greenwald[3] that these lesions are overlooked at operation because of the impression of a normal gallbladder grossly on appearance and palpation, was further illustrated in our case in which the surgeon was reluctant to remove the gallbladder. Further, the pathology department initially missed the papilloma, as it had twisted from its pedicle and lay free in the lumen of the gallbladder.

CASE REPORT

Mrs. L. M. E., a fifty-one year old obese white female was admitted to the gastrointestinal clinic complaining of postprandial epigastric fullness of five months' duration, accentuated by fatty and fried foods, and at infrequent intervals characterized by typical episodes of biliary colic with nausea and emesis.

From the Department of Medicine, Tulane University School of Medicine and the Gastrointestinal Clinic of Touro Infirmary, New Orleans, La.

Physical examination, during an episode of acute manifestation, revealed only right upper abdominal tenderness and muscle splinting.

There were no contributory laboratory findings.

The roentgen findings repeatedly confirmed the presence of a constantly located, marginal, radiolucent shadow as illustrated which was assumed to be a papilloma.

Cholecystectomy was performed by Dr. Irving M. Essrig. Exploration revealed no gross cholecystic disease and cholecystectomy was only performed upon insistence.

The pathologist reported: Benign Glandular Polyp of the Gallbladder.

CONCLUSION

Rarity, roentgen diagnosis, separation of the polyp from the pedicle, obvious potentialities, and the simulation of symptomatology to biliary colic stimulate us to publication of this case.

BIBLIOGRAPHY

1. McHardy, G. & Browne, D. C.: Adenomyoma of the gallbladder. New Orleans M. & S. J., 96:567, 1944.

2. Kirklin, B. R.: Cholecystographic diagnosis of neoplasms of the gallbladder. Am. J. Roentgenol., 29:8, 1933.

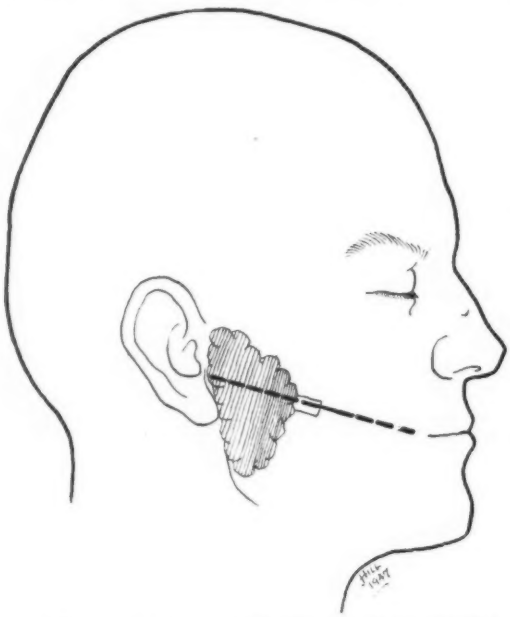
3. Greenwald, W.: Papilloma of the gallbladder. Surgery, 16:370, 1944.

4. Phillips, J. R.: Papilloma of the gallbladder. Am. J. Surg., 21:38, 1933.

REPAIR OF PAROTID DUCT FISTULA
WITH ONE CASE REPORT

LEONARD H. STANDER, M. D.
BATON ROUGE
AND
CLAUDE C. CRAIGHEAD, M. D.†
NEW ORLEANS

The parotid duct, also known as Stensen's or Steno's duct, is approximately five centimeters in length and lies one finger's breadth below the zygomatic arch. It emerges from the extreme anterior portion of the gland and runs forward obliquely on the masseter muscle to the mucous membrane of the cheek. The course of the duct on the masseter muscle is represented by a line drawn from the lower margin of the concha of the ear to the commissure of the lips. The duct opens into the mouth opposite the second upper molar tooth.



The repair of division of the parotid duct may be considered from the standpoint of injuries which are recognized at the time of occurrence and those which are not detected until later. The results are uniformly good in acute injuries with primary repair. However, when injury to the parotid is not detected at initial examination and followed by suturing of the laceration in that region, the repair becomes more difficult.

In primary repair the severed duct can be anastomosed, end to end, after the introduction of some type of inlying dowel which is brought through the oral orifice and fixed to the buccal membrane by a single suture. The dowel should be left in place approximately three weeks. One strand of catgut or silkworm gut is considered satisfactory to keep the duct open. The suture line should not include the mucous membrane of the duct as this predisposes infection.

The most difficult problem encountered is that of a patient with a persistent salivary fistula draining through the scar on the cheek. It is a well recognized fact that an abnormal opening usually closes if the normal channel has an adequate pathway.

Various modifications of von Laugenbeck's[1] technic have been used successfully for secondary repair. Braun[1] employs a mucous membrane tube, Smith a split thickness of skin over a glass rod to reestablish an epithelial lined tract. Glascock[2] threads a piece of silkworm gut through each end of the duct, carries the gut through the gland and attaches a small shot on either end of the suture—one at its exit through the skin, another at its emergence from the mucous membrane.

For many years surgeons have used what is known as a "Seton" suture[3] to produce an internal fistula proximal to severance of the duct. This is accomplished by passing one non-absorbable suture around the duct from within the buccal cavity, tying the suture loosely, and each day pulling the suture slightly until it works through, thereby producing an internal fistula which influences the external fistula to close. However, in this method of treatment the duct papilla with its valve-like action is lost. Newman and Seabrook[4] state that it is essential that the papilla be preserved because of its protective action, its presence being a bar to ascending infection.

If there is any evidence of inflammation present in the cheek laceration it is com-

†From the Charity Hospital of Louisiana, New Orleans, La.

pletely reduced by the use of local heat and penicillin before the institution of operative procedures. The anastomosed ends of the duct have a much better chance of primary union if the wound is clean. Preoperatively a small probe is introduced daily into the buccal opening of Stensen's duct for the purpose of keeping the distal portion of the severed duct dilated thereby enhancing the possibilities of its postoperative function. There must be no stricture of the distal end of the duct if the fistula is to remain closed after operative treatment. The patency of the proximal and distal ends of the duct can be readily ascertained by lipoidal sialography.[5]

Endotracheal intubation is the anesthesia of choice. Local analgesia is contraindicated because the fistulous tract is a potentially infected area. Initially the old scar is completely excised and the severed ends of the parotid duct are dissected free. A ureteral catheter is passed through the distal end of the duct and brought out through the buccal orifice; the other end is threaded into the proximal end of the duct. The proximal and distal ends of the duct are then anastomosed over the catheter using fine interrupted sutures which are passed through the fascial layer of the duct. On the operative side, the catheter is fixed to the commissure of the lip with a single suture and the excess catheter is clipped off. Interrupted sutures are used for closure of the platysma and skin and a pressure dressing is applied.

Penicillin is recommended postoperatively but it is not considered necessary. The catheter is left in position for three weeks. The patient is put on a regular diet, supplemented by pilocarpine and ammonium chloride, to stimulate a free flow of parotid secretion. The small opening in the catheter is kept patent to facilitate drainage. In the event of plugging the catheter is gently twisted. It is our opinion that drainage through the lumen of the catheter accounts for the success of this procedure. This contention, however, is disputed in the literature wherein it is stated that the lumen of the catheter is not essential and further that it does not provide a real channel.[6]

CASE REPORT

E. P., a colored male, aged 33, at the time of admission had the chief complaint of swelling of the right side of the face.

Two weeks prior to examination the patient sustained a cut on the right side of the face inflicted with a pocket knife. An hour later the skin laceration was sutured by his physician. Five days later the sutures were removed and he began to eat solid food again. The right side of his jaw commenced to swell. He returned to his doctor who prescribed sulfa tablets and "rest pills." The patient soon learned that if he "picked the scab off" the wound on his right cheek clear yellow fluid would drain, the swelling would subside, and the pain would disappear. However, as soon as he began to eat again, the mass recurred.

Physical examination revealed a swelling of the right side of the face in the region of the parotid gland. A scar extended from just below the concha of the right ear forward onto the hollow of the cheek. There was a small area on the anterior portion of the scar covered by an encrustation which, when removed, allowed clear yellow fluid to escape.

The patient was operated upon under endotracheal anesthesia. The severed ends of the parotid duct were dissected free and anastomosed over a ureteral catheter which was brought out through the oral orifice of the duct, and fixed to the commissure of the lip with a single suture. The wound was closed in layers, using fine interrupted nonabsorbable sutures. A pressure dressing was applied.

The patient was allowed to eat the first postoperative day. The ureteral catheter was left in place for three weeks. A good functional result was obtained.

The patient was last seen six months postoperatively. At that time he stated that he had experienced no swelling of the jaw following his operation.

SUMMARY

1. A better functional result of the parotid duct is achieved when it is repaired at the time of injury.

2. If the duct is not repaired at the time of original injury, no repair should be attempted until the laceration of the cheek is well healed.

3. Preoperatively the duct should be kept open by daily probing, and postoperatively by some type of inlying dowel, preferably a ureteral catheter.

4. A good functional result is more difficut to attain once a fistula has developed.

5. A typical case is reported.

REFERENCES

1. Manual of Standard Practice of Plastic and Maxillofacial Surgery, W. B. Saunders Co., Philadelphia, Pa., 1942, p. 124.

2. Glascock, H., and Glascock, H., Jr.: Repair of traumatic fistulas of Stensen's duct, Surg. Gynec. & Obst., 65:355, 1937.

3. DaCosta, J. D.: Textbook of Modern Surgery, W. B. Saunders Co., Philadelphia, Pa., 10th ed., 1931, pp. 838-839.

4. Newman, S. C., and Seabrook, D. B.: Management of injuries to Stensen's duct, Ann. Surg., 124:544, 1946.

5. Blady, J. V., and Hocker, A. F.: The application of sialography in non-neoplastic diseases of the parotid gland, Radiology, 32:131, 1939.

6. Butler, E., and Guinan, E. R.: Successful repair of Stensen's duct, Surg. Clin. N. A., 13:1291, 1933.

7. Christofferson, E. A., Ajalat, M. P., and Gradman, R.: Successful primary repair of lacerated parotid duct, Am. J. Surg., 59:592, 1943.

8. Figi, F. A.: Carcinoma of the antrum; transplantation of the parotid (Stensen's) duct, Proc. Staff Meet., Mayo Clinic, 11:241, 1936.

9. Wallace, F. T.: Primary repair of the parotid duct, Am. J. Surg., 70:412, 1945.

10. Wellborn, M. B.: Traumatic fistula of Stensen's duct, South. Surg., 10:793, 1941.

NEW ORLEANS
Medical and Surgical Journal

Established 1844

Published by the Louisiana State Medical Society under the jurisdiction of the following named Journal Committee:

G. C. Anderson, M. D., Ex officio
C. Grenes Cole, M. D., Chairman
E. L. Leckert, M. D., Vice-Chairman
Sam Hobson, M. D., Secretary
C. M. Horton, M. D.
Leon J. Menville, M. D.

EDITORIAL STAFF

Philip H. Jones, M. D.....*Editor*

COLLABORATORS—COUNCILORS

Edwin L. Zander, M. D.
J. T. O'Ferrall, M. D.*
Guy R. Jones, M. D.
T. B. Tooke, M. D.
John Snelling, M. D.
W. E. Barker, Jr., M. D.
C. A. Martin, M. D.
O. B. Owens, M. D.

Paul T. Talbot, M. D.....*General Manager*

1430 Tulane Avenue

* Deceased.

SUBSCRIPTION TERMS: $3.00 per year in advance, postage paid, for the United States; $3.50 per year for all foreign countries belonging to the Postal Union.

News material for publication should be received not later than the eighteenth of the month preceding publication. Orders for reprints must be sent in duplicate when returning galley proof.

Manuscripts should be addressed to the Editor, 1430 Tulane Ave., New Orleans, La.

THE JOURNAL *does not hold itself responsible for statements made by any contributor.*

FEDERAL SUBSIDY OF PEDIATRIC EDUCATION

Federal subsidy of medical education increases the risk of state medicine.

A recent action by the Executive Board of the American Academy of Pediatrics gives organized medicine some cause for concern.

On February 8, 1948, the Executive Board of the American Academy of Pediatrics accepted a report from its committee for the improvement of child health. The sense of this report was somewhat to the following effect: That there were not enough well trained doctors in America to care for the child population. Inference is made that the majority of doctors caring for children are not competent to do so because they have not had hospital training in pediatrics. It is also stated that the doctors are poorly distributed; and further, that the usual channels of pediatric education do not have sufficient money to properly train all the pediatricians needed to take care of America's children. To meet what is described as an urgent situation, this report calls for Federal subsidy of pediatric education. This is to be accomplished by means of grants in aid to medical schools. Congress is being asked for five million dollars to be administered by the Federal Security Administration in direct support of pediatric education. This money is to be used in various ways, such as scholarships or fellowships, or expenses for the staff of pediatric departments when visiting affiliated or outlying hospitals for teaching ward rounds, support for maintaining high standards of pediatric education, and other purposes directly related to pediatric education.

In keeping with such a plan, the Bill was introduced by Senator Thomas of Utah on April 30, 1948. It became Senate Bill No. 2588. Interested parties have appeared in support of this Bill.

Although these plans which are indicated above have passed the Executive Board of the American Academy of Pediatrics, it does not appear, so far, that the Academy as a whole has accepted them. On the contrary, there are some indications that many pediatricians feel that this would be quite inimical to the interests of organized medicine.

This plan of Federal subsidy of pediatrics, if allowed to operate even a few years, would in effect be pediatric State Medicine. If State Medicine is introduced insidiously in this fashion, the difficulty in fighting it in the other fields of medicine is only too obvious. As has been pointed out here before, one has only to look at the extraordinary growth of power of bureaucracy in regard to income taxes to realize how completely dominant so small a thing as a Gov-

ernmental bureau can become in the lives of the entire nation.

Recent rulings of the Supreme Court of the United States indicate that it is the present trend to require participation by negroes in all phases of State and Federal educational policy. The effect of adherence to such provisions in the South would, in many respects, add a great embarrassment even to the problem of State Medicine in the South.

Accordingly, those who support the policies and interests of organized medicine are urged to utilize their influence in opposition to Senate Bill No. 2588 and prevent, if possible, the suckling infant of socialized medicine being wheeled into our midst in a baby carriage.

———o———

TREATMENT OF HYPERTENSION WITH RICE DIET

In 1944, Kempner presented a rice diet for hypertension. The diet contains in 2000 calories not more than 5 grams of fat, and 20 grams of protein, which is derived from rice and fruit, and not more than 200 mgs. of chloride, and 150 mgm. of sodium. The patient takes an average of 250 to 350 grams of rice a day. No sodium chloride or milk is added. If sodium concentration of plain water is greater than 20 milligrams per liter, distilled water should be used. All fruits and fruit juices are used, but not nuts, dates, and avocados; not more than one banana a day; white sugar may be used as desired. Tomato and vegetable juices are not allowed. Supplementary vitamins are added to the diet in the usual form.

The author advocated the rice diet in all serious instances of acute and chronic nephritis and in hypertensive disease. The subjective state of the patients on the rice diet was reported as much improved. Objective findings are as follows: Five hundred patients were on the rice diet for a period of four days to eight hundred and ninety-eight days. The diet was ineffective in 178, including 26 who died. In 322 of the 500 patients, the diet was beneficial, that is, its use was followed by either a reduction in mean arterial blood pressure of at least 20 millimeters, or a reduction of heart size with a change in the transverse diameter of 18 per cent or more, a change in T-1 from a complete inversion to upright, or disappearance of severe retinopathy.

Dick and Schwartz, from the Department of Medicine of the University of Chicago, tested the effect of the rice diet on experimental hypertension in dogs. In this experiment, at the end of eight weeks on the diet, the average arterial blood pressure of the 12 dogs had fallen from 181.6 mm. of mercury to 138 mm. of mercury. In certain dogs, the reduction was as much as 88 per cent. All dogs lost weight on the rice diet, the average being 20 per cent. Grollman and his associates studied the effect of the rice diet with a view to determining the possible value of the absence of sodium in the diet. For this purpose, 100 patients with essential hypertension were placed on a diet, adequate in other respects, but lacking in sodium. Periods of observation varied from several weeks to twelve months. There was a lowering of blood pressure to approximately 155/95 in 20 per cent, and a lowering of the diastolic pressure to, or below, 95 in an additional 15 per cent.

Impression gained from consideration of these several investigations is that dietary treatment of hypertension needs further study. While some of its effects may be due to lack, or sharp reduction in the total amount of sodium consumed, certain other benefits appear to be due to the rice and the fruit. A diet as rigid as Kempner has outlined is one which would be very difficult for the majority of hypertensive patients. On the other hand, when complications of a certain grade of severity appear, the interest of the patient in maintaining the diet will be in proportion to the severity of those complications. It is suggested in a certain portion of these studies that a metabolism of nitrogen and salt is different on the rice diet from what it would be expected to be on a purely fasting diet.

A new field of speculation and interest in hypertension has thus been opened.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

NATIONAL HEALTH ASSEMBLY
WASHINGTON, D. C.
MAY 1-4, 1948

The Secretary-Treasurer of the Louisiana State Medical Society was appointed by the President of the Society to attend this conference as a representative of the organization. Efforts were made to secure a formal delegate's invitation, by writing to Oscar Ewing, Administrator of the Federal Security Administration, however this was unsuccessful and a reply to the request sent to Mr. Ewing was not received. After arriving in Washington every effort possible was made to secure a delegate's invitation to no avail. A reporter's pass was secured and the State Society was thereby represented by the Secretary-Treasurer as a reporter for the New Orleans Medical and Surgical Journal. This proved to be very serviceable as it afforded the privilege of going from one panel meeting to another without interference. The delegates were assigned to one special panel and were unable to get this changed unless request was made for such a change. This press card also served another useful purpose in making available copies of addresses made before all sessions and final reports and recommendations and various other available releases. All of this material has been assembled in book form and is in the office of the Society for ready reference.

On the whole it is felt that the meeting was a successful one especially due to the fact that the 80 doctors in attendance as delegates were able to keep out of the reports the adoption of compulsory health insurance. It was common belief that this conference had been called for the purpose of endorsing this form of national insurance.

The voluntary prepayment plans became more understandable to the public in the manner in which they were presented and discussed. It is believed that the societies sponsoring these voluntary plans gained a lot of friends and supporters as a result of the manner in which they conducted their presentations.

Outstanding in the reports from most of the panels emphasized the fact that the federal government in subsidizing or advancing money for any federal purpose in any state should require that such money be spent on such projects without segregation and no discrimination on account of race, color, creed or religion.

The whole meeting seemed to be packed with a designated, specific purpose, namely, for endorsing federalization of medicine. There were assembled some eight or nine hundred individuals; a very cosmopolitan group composed of scientists, doctors, researchers, labor people, welfare workers and most important of all, most of those people were from the federal government and principally from the Federal Security Administration. They seemed to have priority of receipt of invitations. Some of the speakers who represented labor and other groups could hardly speak English. The renowned Dr. Mike Davis made a statement that "the more the AMA opposes federal health insurance the more surely will the country be driven into state medicine".

Throughout the meeting in the section on medical care, it was quite common to see lobbying by various types of people, moving around amongst the audience prompting and conversing quietly with their followers, evidently preparing some strategic action on their part. The audience in the medical care section at times broke away from what would ordinarily be considered good decorum for such a gathering. For example when a favored speaker would make a point in his discussion which they approved they would clap and holler and cheer and when

the opposition spoke and made some remark with which they did not agree they did not hesitate to boo. Sometimes it was felt that if one walked into a communistic meeting or a labor union gathering the setup in this regard could not have been more idealistic.

One of the most outstanding observations on the part of the representative of the State Society was that with all of this the representatives of the medical profession did not lose their heads and were complimented several times on the manner in which they conducted their work and presented their cause.

Following is further information concerning this Assembly, as reported by Dr. Jos. S. Lawrence, Director of the Council on Medical Service of the American Medical Association.

"The National Health Assembly began its sessions on Saturday morning, May 1st, with more than eight hundred delegates registered of whom approximately one-fourth were physicians, the others being members of State Health Departments, social workers, representatives of labor unions, farm groups, consumers, and civilian organizations. At the opening session the delegates were welcomed by Honorable Oscar R. Ewing, Federal Security Administrator; and Howard M. Kline, the Executive Secretary, announced that the delegates would be registered into fourteen divisions, each with its own room, and most of them located in the Statler Hotel where the headquarters of the Assembly were established.

The machinery of the Assembly consisted of an Executive Committee appointed by the Administrator more than a month ago and a chairman and steering committee for each division. The Executive Committee advised the Administrator on the details of the general program and, at the close of the Assembly, will consider with him the reports submitted by the division chairmen. The steering committees of the divisions prepared in advance statements suggesting subjects for discussion by the members of the division. At the opening meetings of the divisions, these statements were read or delivered, after which open discussion was entertained."

Membership in the divisions ranged from seventy-five upward, the largest division being that of "Medical Care" in which several hundred were registered. A list of the sections follows:

1. What is the Nation's Need for Health and Medical Personnel?
2. What is the Nation's Need for Hospital Facilities, Health Centers and Diagnostic Clinics?
3. What is the Nation's Need for Local Health Units?
4. Chronic Disease and the Aging Process.
5. A National Program for Maternal and Child Health.
6. A National Program for Rural Health.
7. What is the Nation's Need for Research in the Service of Health?
8. What is the Nation's Need for Medical Care?
9. State and Community Planning for Health.
10. Physical Medicine and Rehabilitation.
11. What Can Be Done to Improve Dental Health?
12. A National Program for Mental Health.
13. What Can Be Done to Improve Nutrition?
14. A National Program of Environmental Sanitation.

"The divisions were in session Saturday afternoon, Sunday morning and afternoon, and Monday afternoon. Several general sessions were held which were open to the public. On Saturday evening there was a dinner for the delegates and their friends which was addressed by President Truman. There were also two special luncheons.

The findings of the various divisions will be published in time. The recommendations and reports of the committees of the division were not voted upon by the members of the committees. No efforts were made in any division to record opinion on any matter that was under discussion. I was

not registered as a delegate to any division, but with the assistance of a press card I was able to visit all the divisions while they were in action. The general opinion is that the committees took their work very seriously. Two objectives seemed to present themselves in one form or another to each group: (1) that the problem assigned to it is a community problem and (2) that its solution is the responsibility of all of the agencies of the community.

In the most instances, of course, the subject under discussion related to health, but the delegates recognized that the problem could not be solved by physicians and health authorities alone. *In only two or three instances did the committees emphasize the need of Federal assistance.* Even in the Medical Care Committee *compulsory health insurance gave way to consideration of voluntary plans* as is evidenced by a statement which was submitted by the Steering Committee of the Medical Care Division at its last session.

There was much discussion as to how the prepayment plans should originate and by whom they should be controlled. Representatives of the cooperatives, the consumers, and the unions generally felt that control should not be left to the physicians alone, and opposition was expressed to legislation extant in more than twenty states requiring that no plans be organized without approval of the State Medical Association.

At a general session on Tuesday morning, Quincy Howe, of the Columbia Broadcasting System, reported the findings of all of the divisions and commented that this is probably the first such occasion where laymen and physicians worked together so zealously to develop a health program. All divisions *approached their tasks from the community rather than the national level.* The Assembly closed with a luncheon held in observance of the 150th Anniversary of the U. S. Public Health Service."

—o—

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of every month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

SABINE PARISH MEDICAL SOCIETY

The meeting date of the Sabine Parish Medical Society will be the second Wednesday of each month, and the place will be rotated from Zwolle, to Many, to Pleasant Hill, to Converse. The next meeting will be in Zwolle on June 9 at the home of Dr. L. H. Murdock.

CARDIOLOGY COURSE IN MEXICO CITY

This note under College News appeared in the Annals of Internal Medicine, April 1948, p. 877.

This will be of great interest to Louisiana physicians who are planning to take post graduate work and a vacation in a cool climate at the same time.

A two-week course in cardiology August 2 to 13 will be held at the National Institute of Cardiology in Mexico City under the direction of Dr. Ignacio Chaves, F. A. C. P., with the instructional staff being made up of a number of outstanding Mexican teachers who speak English, supplemented by several distinguished guest teachers from the U. S. Sessions will be held in the mornings daily, with the afternoons free for entertainment, sightseeing and visits to other institutions. This course presents an opportunity for a most enjoy-

able two weeks' vacation, partly devoted to study and observation of methods in our neighboring Republic. Wives and members of the physicians' families are invited, and adequate hotel accommodations will be available. Those interested in this course are requested to communicate at once with the Executive Secretary of the College, Mr. E. R. Loveland, 4200 Pine Street, Philadelphia 4, Pennsylvania, so that some definite estimate can be prepared for the Director and the hotels.

THE AMERICAN CONGRESS OF PHYSICAL MEDICINE

Will hold its twenty-sixth annual scientific and clinical session Sept. 7, 8, 9, 10 and 11 inclusive, at the Hotel Statler, Washington, D. C. Scientific and clinical sessions will be given the days of Sept. 7, 8, 9, 10 and 11. All sessions will be open to members of the medical profession in good standing with the American Medical Association. In addition to the scientific sessions, the annual instruction courses will be held Sept. 7, 8, 9 and 10. These courses will be offered in two groups. One set of ten lectures will be based primarily on physics and physiology and attendance will be limited to physicians. One set of ten lectures will be more general in character and will be open to physicians as well as to physical therapists. The physical therapists must be registered with the American Registry of Physical Therapy Technicians. Full information may be obtained by writing to the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

NAVAL HOSPITAL RESIDENCIES

Rear Admiral Clifford A. Swanson, MC, USN, Surgeon General and Chief of the Bureau of Medicine and Surgery, announces that a new residency in General Practice will be inaugurated in Naval Hospitals on July 1, 1948. The Professional Division of the Bureau of Medicine and Surgery has developed a residency in that phase of medicine which is required to augment the several medical specialties recognized by the American Specialty Boards in order to supplement the residency training program, now operative, which is designed toward specialization. The program, as planned, will cover a period of three years. During each year of this residency Medical Officers will receive six months of training in General Medicine and related specialties, and six months in General Surgery and related specialties. Instruction will be provided by the staffs of the hospitals and outstanding civilian visiting staffs. With the introduction of this type of training Navy medicine has recognized the great need of the General Practitioner in the field of medicine.

MEETING OF L. S. U. MEDICAL ALUMNI

The New Orleans District of the L. S. U. Medical Alumni Association will meet at a banquet at Arnaud's Restaurant on June 10, at 7:30 p. m. Dr. Harold Stoke, President of Louisiana State University will be the principal speaker. Alumni should send $3.50 for their reservations to the Secretary-Treasurer, L. S. U. Medical Alumni Association, L. S. U. Medical School, 1542 Tulane Avenue.

WOMAN'S AUXILIARY

The Woman's Auxiliary to the Louisiana State Medical Society held its annual meeting in conjunction with the 68th annual conference of the Louisiana State Medical Society April 12-14 in Monroe, La. Headquarters for the Auxiliary were in the Virginia Hotel.

Following registration on Monday a luncheon for all state officers and officers of the Ouachita Parish Auxiliary was given at the La Casa Tearoom. A pre-convention Executive Board Meeting was held following the luncheon with Mrs. James W. Warren presiding. Reports of chairmen were read.

The home of Dr. and Mrs. DeWitt Milam was the lovely setting for a tea Monday afternoon, when the members of the Ouachita Auxiliary honored the following: Mrs. Eustace Allen, President of the Woman's Auxiliary to the American Medical Association, Mrs. Olin S. Cofer, President, Woman's Auxiliary to the Southern Medical Association, Mrs. A. A. Herold, Treasurer, Woman's Auxiliary to the American Medical Association, Mrs. O. B. Owens, President-elect of the Woman's Auxiliary to the Louisiana State Medical Society, and Mrs. James W. Warren, President of the Woman's Auxiliary to the Louisiana State Medical Society.

Tuesday, the General Session of the Auxiliary was held in the Crystal Ball Room of the Virginia Hotel with the President, Mrs. James W. Warren of New Orleans presiding. The invocation was given by Msgr. J. C. Marsh, St. Matthews Catholic Church. The address of welcome was given by Mrs. J. E. Walsworth, President of the Ouachita Parish Auxiliary and the response by Mrs. T. E. Strain, President, Caddo Parish Auxiliary. Memorial Services were read by Mrs. A. Dent Tisdale and the benediction, a vocal solo, "The Lord's Prayer" by Mrs. A. M. Serex.

Greetings were given to the assembly from Dr. Gilbert Anderson, New Orleans, President 1947-48 of the State Society, Dr. Marion Hargrove, Shreveport, President-elect 1948-49, Dr. John G. Snelling, Monroe, Chairman of Convention, and Dr. A. V. Friedrichs, Chairman of Council on Medical Service and Public Relations. This Council has offered their facilities for printing and mailing a Quarterly Publication for the Auxiliary.

Mrs. Eustace A. Allen, President of the Woman's Auxiliary to the American Medical Association, brought greetings from National. Their aim is to double the membership of the Auxiliary. There are 130,000 doctors who belong to the A. M.

A. and only 35,000 wives to the Auxiliary. She complimented Louisiana and the South on their progressive attitude towards a broad Public Relations program and she further stated that we as individuals should study the Bulletin, Hygeia, AMA and State Journals and know our subject matter in order to aid our husbands in the "art of letting the public know what they are doing for their good."

Mrs. Olin S. Cofer, President of the Woman's Auxiliary to the Southern Medical Association, was introduced as an honored guest and brought us a splendid message from "Southern." The business meeting was adjourned until the afternoon. The doctors and their wives were entertained at a barbecue arranged by Dr. D. W. Milam and his Committee. At the afternoon session reports were given by the State Officers and Parish Presidents. A report on the A. M. A. convention was read from Mrs. George Taquino. Mrs. Arthur A. Herold gave a report on the Southern Medical Association Convention. Mrs. M. C. Wiginton gave a report as our representative to the Louisiana Rural Health Council.

After the transaction of old and new business election of officers was held followed by their introduction and installation by Mrs. H. B. Gessner. The incoming President announced that the Post-Convention and School of Instruction to be held Wednesday morning would be open to all delegates and guests.

A complimentary dinner-dance was held Tuesday evening on the Roof of the Virginia Hotel. Wednesday a luncheon and style show followed the meeting which climaxed three days of pleasure for members of the Auxiliary in attendance at the State Convention. Members of the Ouachita Parish Auxiliary distinguished themselves as "perfect hostesses" throughout the Convention. The following who served on special committees should especially be complimented: Mrs. DeWitt Milam, General Chairman; Mrs. J. E. Walsworth, President and Vice-Chairman; Chairmen of Committees: Mrs. Clifford U. Johnson, Registration; Mrs. C. P. Gray, Jr., Badge and Ticket; Mrs. Morgan Simonton, Supplies; Mrs. A. Dent Tisdale, Program; Mrs. P. L. Perot, Information; Mrs. J. W. Cummins, Transportation; Mrs. C. B. Flinn, Publicity; Mrs. A. Scott Hamilton, Luncheon; Mrs. H. V. Collins, Decorations; Mrs. Irving Wolff, Tea.

Mrs. F. U. Darby,
Press and Publicity Chairman of
The Woman's Auxiliary to the
La. State Medical Society.

Following is the President's Annual Report for 1947-48.

As President of the Woman's Auxiliary to the Louisiana State Medical Society, I beg leave to submit the following report:

The Louisiana Auxiliary brings greetings and a warm welcome to our National President, Mrs. Eustace Allen; to our Southern President, Mrs. Olin Cofer; to our National Treasurer, Mrs. Arthur Herold, who are our honored guests. The Auxiliary feels that Louisiana has made a distinct contribution to the National Association this year in supplying a treasurer so capable as our own Mrs. Herold. We know her. We admire her for her ability and love her for herself. We are proud to have her represent us among the outstanding women on the National Board.

The term program as applied to our Auxiliary has come to encompass practically every phase of Auxiliary work, and has gone so far beyond the old idea of entertainment that it requires not only the program chairman to administer it, but the aid of the vice-presidents, councilors, committee chairmen and parish presidents. Remembering that we are liaison officers between doctors and the public, as Mrs. Allen has so well put it, we have acquired a desire to be better informed. So the program, per se, has taken on a more serious form in most Auxiliaries. Some of the many fine program topics that have been offered follow: Community Recreation; Medical Legislation in the 80th Congress; Social Problems of To-day; Status of Tuberculosis Control in Louisiana; Juvenile Delinquency; Early Diagnosis of Cancer, Trends in Nutrition.

Blood and Plasma Banks are sponsored by two Auxiliaries. Nearly every group reports work or donations to Red Cross, March of Dimes, and T. B. Seals. Wives of Lafourche doctors helped their husbands in the examination of pre-school children and 4H clubs. Such activities demonstrate a general awareness of social needs and community welfare.

In promoting the most vital project on our agenda, The Extension of Medical Services through Louisiana Physicians' Service, the Public Relations chairman has been of inestimable help, as well as in other phases of the general program. Radio programs, sponsored by Parish Medical Societies are being put on with the help of Auxiliaries. Monroe and Shreveport have active speakers bureaus, and when called on, send speakers to any organized club to talk on Voluntary Prepayment plans. Caddo, Rapides and East Baton Rouge had open meetings, well attended by the laity, at which facts about Louisiana Physicians' Service were presented. Guests were also told of the stupendous sums—$118,000,000 in 1947—that the Federal government, through various agencies, has expended on propaganda for compulsory health insurance.

We have become keenly conscious of the need for improved health and medical service in rural areas, and our group has an active representative on the Louisiana Rural Health Council.

Louisiana has sponsored two new projects this

year. The first, Preservation of Medical Cultural Items, is designed to collect and preserve historical medical data. The State's medical history is especially rich, but unfortunately too many early records and documents have been lost in Louisiana, and an effort is now being made to save what is left. We are indebted to Mr. William D. Postell, Librarian of the State University Medical School, for stimulating an interest in this work by his enlightening addresses, delivered to a number of Auxiliaries.

Calcasieu has compiled a history of the early doctors of the parish; they have had good publicity on the subject with an editorial and a front page article in the local paper. A window display featured on Doctors' Day showed scrap books, pictures of old instruments, pictures of an early drug store. East Baton Rouge featured a history of doctors of the parish from 1880 to 1926. Shreveport had editorials and a full page of pictures of items of interest. A member of the Jefferson Davis group has compiled a history of the early doctors of the parish, and books and instruments are to be turned over to them soon.

The Second District has books, one published in 1834, a Fitch Scale, scientific papers. Iberville has obtained old scales, books, a cautery set, a baby resuscitater, pictures of older doctors and a copy of the minutes of the first meeting of the Parish Medical Society. Rapides, Ouachita and Orleans have appointed chairmen—the latter has a few instruments—and five parishes have had addresses on the subject. The outlook is encouraging.

While we have had no chairman for Student Nurse Recruitment, the second new project, a number of parishes and the Second District have been active. They are sponsoring talks to high school classes, having essays written, displaying posters and aiding local hospitals in their efforts to obtain students. Mrs. Donaldson of Second District, secured pledges from eleven girls to study nursing. We recognize the crucial need for nurses in both rural and urban areas and it behooves us to spend more effort to attract them.

A definite consciousness of the need to stress cancer control is shown in every parish report received. Activities include making dressings, having lectures, sponsoring essays, gifts of money, work in the April drive, presentation of films and radio programs, not only to Auxiliaries but to schools, parent-teacher groups and women's clubs. Members in many parishes serve as commanders, deputies, and campaign chairmen.

Doctors' wives in every parish have honored their husbands on their Day—March 30th. Some in the traditional manner, with parties; others by featuring exhibits of medical cultural items; some with donations to the Commemoration Fund. Gifts to this fund, for aid to needy widows of doctors, have totaled $221.04, the largest for any one year so far, bringing the total amount in the bank to $780.79.

Our desire to have better informed members who are more conversant with topics of special interest to us as wives of doctors, is gratified by the report of the Bulletin Chairman. She reports that subscriptions have increased from 23 last September to 82 at the present time, a gain of 59. It is sincerely hoped that interest in the Bulletin may continue. I believe that it is not a mistake to say that programs in all lines of our work may be directly due to this wider use of the publication.

The chairman of Press and Publicity has each month supplied the State Journal with interesting material for the Auxiliary section. She has kept readers informed concerning the program and the accomplishments of the parish groups. The files of last year have been placed in the Archives. The historian has compiled an attractive book, not, however, without some difficulty. Some parishes have neglected to send her any material at all, and others have not sent as much as they should. Please keep this in mind another year and remember that the historian needs your help in order to keep a full record of Auxiliary accomplishments.

Several parishes have done outstanding work in securing subscriptions to Hygeia, notably Ouachita, home of the Chairman, where eighty subscriptions were obtained. Tangipahoa has an excellent record, with every member a subscriber, and Iberville has placed subscriptions in every school. Jefferson Davis, with only eleven members, has five readers of Hygeia, four of the Bulletin, nine each of the New Orleans and A. M. A. Journals. They, surely, are among our best informed members.

With the President of the United States, in a recent speech, openly favoring compulsory medical insurance, the State Medical Society supporting certain parts of the Taft Health Bill, and Congress reconvening in January, a definite impetus was given to interest in legislation. The chairman has supplied parishes with material from News Letters, A. M. A. Journals, pamphlets and other sources. A few groups, though not enough, have devoted programs to legislation.

There was no need for revision of by-laws this year, but your chairman has stood by, ready to be called on.

Two distinct time-saving services were rendered officers and members when the printing chairman had names and addresses of parish presidents placed on the stationery, and the year-book chairman included the addresses of all members in the larger towns and cities in the year-book.

Progress has been made in organization, with Vernon Parish, with eight members, joining our ranks. Washington, with fifteen members, has organized and elected officers. A four-parish group, with Ruston as the center, came into being in November, but shortly afterwards, due to illness of the leader, became inactive. Lafayette has again

become active, and we have the definite promise that St. Landry will again join us in the fall. It has been especially inspiring to see the large numbers of fine young women who have become members in all sections of the State. This argues well for the future of the Auxiliary. There is a total of 786 full paid members, 18 associate and 10 honorary, making a grand total of 812.

At the suggestion of the President-elect, Mrs. Owens, a committee was appointed at the February Board meeting to look into the matter of having an Auxiliary Publication. As a result, the State Council on Medical Services have offered their facilities for printing and mailing a Quarterly publication for the Auxiliary. This will be invaluable in bringing direct to the doctor's wife information she should have, increasing thereby the effectiveness of the Auxiliary program.

Our National officers have been a source of inspiration and encouragement throughout the year. The executive secretary, Miss Wolfe has been helpful and co-operative. My own Board and the membership as a whole have given me such support as few presidents even hope for. To all of you I am deeply grateful for a happy, albeit strenuous year.

May I also express sincere appreciation to the President and Executive Secretary of the Louisiana State Medical Society, Dr. Anderson and Dr. Talbot, and to Miss Shoemaker, their assistant; and to the members of the Advisory Board, Dr. Shirley Lyons, Dr. Charles Barker, and Dr. Rhodes Spedale, for their unfailing courtesy and assistance in solving Auxiliary problems.

Respectfully submitted,

Barbara P. Warren, President,
Woman's Auxiliary to the Louisiana
State Medical Society.

BOOK REVIEWS

Practical Office Gynecology: By Karl John Karnaky, M. D. Springfield, Charles C. Thomas, 1947. Pp. 261. Price, $7.50.

One is immediately impressed by the personal nature of this book and the varied indications advocated for stilbestrol. Nausea is encountered in 87 per cent of 3223 patients receiving stilbestrol but this disappeared in 94 per cent after four or five days of treatment.

The author prescribes stilbestrol for the various menstrual dysfunctions, threatened abortion, preoperatively in control of hemorrhage, uterine bleeding in the presence of myomas, chronic salpingitis, et cetera.

It is repeatedly emphasized that where malignancy is suspected, a thorough curettage and examination for cancer is essential.

Karnaky deserves due credit for his enthusiastic and persistent use of stilbestrol, for he has been bold where others fear to tread. Much of the experimentation needs substantiating, as stilbestrol is no panacea.

The compilation of this office reference book is excellent and one will find a treatment definitely outlined for almost every gynecologic ailment. Dr. Louis Spivak contributes a short chapter on some psychological aspects of gynecology.

EUGENE H. COUNTISS, M. D.

Ocular Therapeutics: By Wm. J. Harrison, Phar. D., M. D., F. A. C. S., Springfield, Charles C. Thomas, publisher, 1947. Pp. 112. Price, $3.50.

This pocket manual of approximately one hundred pages briefly presents a description of the properties, action and uses of the more frequent drugs employed in ophthalmology. The use of moist heat and cold, buffer and isotonic solutions, and local anaesthetics are also briefly discussed in a very readable and understandable manner. Metric prescriptions accompany most of the drugs mentioned. Younger ophthalmologists and those who are employed in ophthalmic clinics and offices will find much of value in this small volume on which the publisher has also done an excellent job.

CHAS. A. BAHN, M. D.

Gifford's Ocular Therapeutics, 4th. Edition: By D. Vail, Philadelphia, Lea & Febiger, 1947. Pp. 336. Price, $5.00.

Dr. Vail has rendered a needed service in revising this well known work. The current usages of miotics, antibiotics, vascular therapy, as well as thermo and electro therapy are discussed at some length. On the whole, the changes which have been made were well chosen and the literature thoroughly reviewed. Books on therapeutics are especially difficult to present understandingly. They are written on the assumption that the remedies suggested will be employed in patients whose ocular symptoms and disease have been completely and accurately diagnosed. Unfortunately, this is often not correct. In subsequent editions the author should consider greater coordination of local and bodily therapy. Rarer and less remedial ocular conditions are occasionally discussed at greater length than their practical importance apparently justifies in a work of this size. The basic principles involved in the care of frequent ophthalmic conditions such as the allergic or/and psychoso-

matic factors in ophthalmic diseases, as well as seborrheic blepharoconjunctivitis, should be explained at greater length for greater practicality. Especially for younger ophthalmologists and those not familiar with current literature, this volume is a must.

CHAS. A. BAHN, M. D.

Osteotomy of the Long Bones: By Henry Milch, M. D. Springfield, Charles C. Thomas, 1947. Pp. 294. Price, $6.75.

Osteotomy of the Long Bones by Henry Milch is a readable, well organized, and short book. It appears to be designed to impart to the specialist or the serious student an understanding of the fundamental principles underlying osteotomies. It is not intended to show the novice how to do them.

Dr. Milch develops, step by step, one's understanding of the problems involved in osteotomies; from the maintenance of parallelism of the articular ends in the simple angular osteotomy, to the more complex relationships in multiple planes, which are involved in osteotomies about the hip. For that reason, this is a book to be read through and savored, rather than one to be skipped about in.

There are many practical and worth while features in this book that deserve commendation, among them are: the clear and complete discussion of treatment of inequalities of limb length, the detection and correction of torsional deformities, and the methods of obtaining either increased stability or increased range of motion in the pathological hip.

ARTHUR MAXWELL, M. D.

PUBLICATIONS RECEIVED

Blakiston Company, Philadelphia: Human Physiology (3rd edit.), by F. R. Winton, M. D., D. Sc. and L. E. Bayliss, Ph. D.

Lea & Febiger, Philadelphia: Laboratory Diagnosis of Protozoan Diseases (2nd edit.), by Charles Franklin Craig, M. D., M. A. (Hon.), D. Sc. (Hon.), F. A. C. S., F. A. C. P.

Medical Society of the County of Westchester, White Plains, New York: History of the Medical Society of the County of Westchester 1797-1947.

C. V. Mosby Company, St. Louis: Neuroanatomy (2nd edit.), by Fred A. Mettler, A. M., M. D., Ph. D.; Synopsis of Pediatrics, by John Zahorsky, A. B., M. D., F. A. C. P.

Charles C. Thomas, Springfield, Illinois: Coronary Heart Disease, by A. Carlton Ernstene, M. D.; The Hospital Care of Neurosurgical Patients (2nd edit.), by Wallace B. Hamby, M. D., F. A. C. S.

Williams and Wilkins Company, Baltimore: The Biological Standardisation of the Vitamins (2nd edit.), by Katharine H. Coward, D. Sc.; Psychiatric Examinations of the School Child by Muriel Barton Hall, M. D.; Text-Book of Public Health (12th edit.), by W. M. Frazer, O. B. E., M. D., Ch. B., M. Sc., D. P. H. and C. O. Stallybrass, M. D., Ch. B., D. P. H., M. R. C. S., L. R. C. P.

World Press, Inc., Denver: War, Politics, and Insanity, by C. S. Bluemel, M. A., M. D., F. A. C. P., M. R. C. S. (Eng.).

INDEX TO VOLUME ONE HUNDRED

July, 1947 — June, 1948

—A—

Acute Mastoiditis—Secondary to Fracture of the Mastoid, by Clifford Vedrenne and Jack R. Hays 504
Adult Hypothyroidism and Myxedema, by Norman Burnstein 374
Airblock in the Newborn Period, by George W. Salmon 253
Akenhead, Walter R., Extra-Intestinal Amebiasis 105
Alcoholic, Clinical Observations on the Treatment of the, by Roy Carl Young 539
Altenberg, Alfons R., Double Notched Graft for Lumbosacral Spine Fusion 464
Amebiasis, Extra-intestinal, by Walter R. Akenhead 105
Amebiasis, in Children, Some Recent Experiences with, by Maud Loeber and Joseph S. D'Antoni 276
Anemia, Chronic, The Treatment of, by Norton W. Voorhies 310
Anesthesia, Saddle Block, in Obstetrics, by E. L. King and Isadore Dyer 70
Aneurysm, Myocardial, Causing Esophageal Obstruction, by Morris Isaacson 568
Annual Meeting, Program for 1948 386
Anovulation, A Survey of General and Ovarian Sterility, with Special Reference to, by Stonewall B. Stickney 26
Anti-Amebic Drugs, An Evaluation of, by Kenneth G. Nix 108
Antibiotics and Chemotherapy, Symposium on Chemotherapy and Antibiotics, by Maxwell Finland 345
Anus, Imperforate, Urological Complication Following Operation for, by Robert F. Sharp 456
Appointment of Editor 331
Arey, James B., Postmortem Observations in Twenty-two Premature Infants 258
Atresia, Farber's Test in the Diagnosis of Congenital Intestinal, by Norman C. Woody 273
Auxiliary, Woman's, to the Louisiana State Medical Society 192
Aymond, Branch J., Poliomyelitis 491

—B—

Bain, Lamar, Medical Aspect of 6-Propylthiouracil. (Hull, Bain) 53
BAL in Heavy Metal Poisoning, Drug Reactions and Their Treatment, with Special Reference to the Use of, by C. Barrett Kennedy and V. Medd Henington 209
Barker, H. O., Hypertrophic Pyloric Stenosis. (Hardy, Jr., White, Barker) 307
Biopsy, Needle, of the Liver, by W. D. Davis, Jr. 159
Bodenheimer, J. M., Fractured Ribs from Coughing 474
Book Reviews 51, 92, 139, 188, 251, 293, 342, 395, 444, 488, 534, 585
The "Borrowing-Lending" Hemodynamic Phenomenon (Hemometakinesia) and Its Therapeutic Application in Peripheral Vascular Disturbances, by Thorpe Ray, George E. Burch, and Michael E. DeBakey 6
Bowel, The Large, The Diagnosis and Treatment of Acute Malignant Obstruction of, by Marshall L. Michel 397
Boyce, Frederick Fitzherbert, Thiouracil in Toxic Thyroid Disease. (Dieter, Boyce) 56
Bresler, E. H., The Pathogenesis of the Edema of Heart Failure 297
Brown, Wallace H., Peritoneoscopy. (Hollis, Brown) 498
Browne, E. Zollicoffer, The Use of Curare in Tetanus. (Stone, Browne) 383
Buffington, W. R., Congenital Ocular Anomalies Resulting from (a) Premature Birth (b) Rubella Infection of Mother during Pregnancy 466
Burch, George E., The "Borrowing-Lending" Hemodynamic Phenomenon (Hemometakinesia) and Its Therapeutic Application in Peripheral Vascular Disturbances. (Ray, DeBakey, Burch) 6
Burnstein, Norman, Adult Hypothyroidism and Myxedema 374

—C—

Caldwell, Pearson C., Facial Nerve Paralysis Resulting from Fracture of the Temporal Bone. (Farrior, Caldwell) 23

Cancer Detection Clinic, The Hutchinson Memorial, by Robert L. Pye 72
Cancer, Gynecologic, Radiotherapy for, by Manuel Garcia and Joseph V. Schlosser 141
Cancer, Radiation Treatment in, by Stakely Hatchette 316
Central Fractures of the Neck of the Femur, by I. L. George 459
Chemotherapy and Antibiotics, by Maxwell Finland 345
Chemotherapy and Antibiotics, Symposium on
Maxwell Finland 345
Champ Lyons 358
George W. Salmon 361
Gerald J. Joseph 367
The Chemotherapy of Leprosy, by Herman Gray 218
Chanton, Edwin F., Myxedema. (Hargrove, Chanton) 546
Chromomycosis, by Leslie K. Mundt and Morris Moore 558
Chronic Pneumonias, by Sidney Jacobs 207
Circumvallate Placenta, by Arthur B. Hunt, Robert D. Mussey, and John E. Faber 203
Clinical Observations on the Treatment of the Alcoholic, by Roy Carl Young 539
Clinico-Pathologic Conference, by Charles E. Dunlap and Edgar Hull 324
Congenital Hemolytic Jaundice, by Robert Kapsinow 66
Congenital Ocular Anomalies Resulting from (a) Premature Birth (b) Rubella Infection of Mother during Pregnancy, by W. R. Buffington 466
Congestive Heart Failure, The Pathogenesis of the Edema of, by E. H. Bresler 297
Coughing, Fractured Ribs from, by J. M. Bodenheimer 474
Craighead, Claude C., Repair of Parotid Duct Fistula, (Stander, Craighead) 574
Curare, The Use of in Tetanus, by E. Zollicoffer Browne and Harry A. Stone 383

—D—

D'Antoni, Joseph S., Some Recent Experiences with Amebiasis in Children. (Loeber, D'Antoni) 276
Daron, Paul B., The Etiology of Peptic Ulcer 163
Davenport, J. W., Jr., Human Isoimmunization 376
Davenport, J. W., Jr., The Rh Factor in Clinical Obstetrics and Gynecology 510
Davis, W. D., Jr., Needle Biopsy of the Liver 159
DeBakey, Michael E., The "Borrowing-Lending" Hemodynamic Phenomenon (Hemometakinesia) and Its Therapeutic Application in Peripheral Vascular Disturbances. (Ray, Burch, DeBakey) 6
The Determination of Serum-and Urine-Salicyl Levels during Treatment with Salicyl Drugs, by Johan T. Peters 1
The Diagnosis and Treatment of Acute Malignant Obstruction of the Large Bowel, by Marshall L. Michel 397
Dicoumarin—Medical Aspects, by Thomas Findley 413
Dicumarol, The Use of in the Prevention and Treatment of Intravascular Clotting and Embolism, by Samuel A. Romano 506
Dieter, Donald D., Thiouracil in Toxic Thyroid Disease. (Boyce, Dieter) 56
(Diethyloxide) Intravenous Ether, by O. C. Williams 470
D'Ingianni, Vincent, Rapid Postoperative Mobilization 80
Double Notched Graft for Lumbosacral Spine Fusion, by Alfons R. Altenberg 464
Drugs, Anti-Amebic, An Evaluation of, by Kenneth G. Nix 108
Drug Reactions and Their Treatment, with Special Reference to the Use of BAL in Heavy Metal Poisoning, by C. Barrett Kennedy and V. Medd Henington 209
Duct Fistula, Parotid, Repair of, by Leonard H. Stander and Claude C. Craighead 574
Dunham, Ethel C., Prematurity—An Orientation 119
Dunlap, Charles E., Clinico-Pathologic Conference. (Hull, Dunlap) 324
Dyer, Isadore, Saddle Block Anesthesia in Obstetrics. (King, Dyer) 70

—E—

Editor, Appointment of 331
Editorials 39, 83, 131, 179, 240, 281, 333, 384, 435, 480, 527, 577
Edwards, Edwin, Papilloma of the Gallbladder. (McHardy, Edwards) 573

Embolism, The Use of Dicumarol in the Prevention and Treatment of Intravascular Clotting and, by Samuel A. Romano 506
Encephalomyelitis, Equine; Human Infection with Virus of, Eastern Type, in Louisiana, by Kelsey C. Milner, G. Peyton Kelley, and Morris F. Shaffer 270
Encephalimyelitis, Human Equine, Eastern Type. in Louisiana, by George H. Hauser 551
Enteric Infection or Infestation Sine Dysentery, by Daniel N. Silverman and Alan Leslie 101
Equine Encephalomyelitis, Human Infection with Virus of, Eastern Type, in Louisiana, by Kelsey C. Milner, G. Peyton Kelley and MorrisF. Shaffer 270
Epidemiology and Recent Developments in Poliomyelitis, by Joseph G. Molnar 408
Erickson, Paul T., Sulfone Treatment of Leprosy. (Wolcott, Johansen, Erickson) 475
Esophageal Obstruction, Myocardial Aneurysm Causing, by Morris Isaacson 568
Ether, Intravenous (Diethyloxide), by O. C. Williams 470
The Etiology of Peptic Ulcer, by Paul B. Daron 163
An Evaluation of Anti-Amebic Drugs, by Kenneth G. Nix 108
Extra-Intestinal Amebiasis, by Walter E. Akenhead 105

—F—

Faber, John E., Circumvallate Placenta. (Hunt, Mussey, Faber) 203
Facial Nerve Paralysis Resulting from Fracture of the Temporal Bone, by J. Brown Farrior and Pearson C. Caldwell 23
A Family Outbreak of Lead Poisoning from Storage Battery Casings, by David Spizer 278
Farber's Test in the Diagnosis of Congenital Intestinal Atresia, by Norman C. Woody 273
Farrior, J. Brown, Facial Nerve Paralysis Resulting from Fracture of the Temporal Bone. (Caldwell, Farrior) 23
Farrior, J. Brown, Sigmoid Sinus Thrombosis. (Hart, Farrior) 20
Femur, Central Fractures of the Neck of the, by I. L. George 459
Findley, Thomas, Dicoumarin—Medical Aspects 413
Finland, Maxwell, Chemotherapy and Antibiotics 345
Fistula, Parotid Duct, Repair of, by Leonard H. Stander and Claude C. Craighead 574
Fractured Ribs from Coughing, by J. M. Bodenheimer 474
Frederick, C. O., Hematuria: Its Clinical Significance 517
Functional Conditions of the Nose, by Joseph Stamm 322

—G—

Gallbladder, Papilloma of the, by Gordon McHardy and Edwin Edwards 573
Garcia, Manuel, Radiotherapy for Gynecologic Cancer. (Schlosser, Garcia) 141
George, I. L., Central Fractures of the Neck of the Femur 459
Getzoff, Paul L., Impotence 77
Gray, Herman, The Chemotherapy of Leprosy 218
Gynecologic Cancer, Radiotherapy for, by Manuel Garcia and Joseph V. Schlosser 141

—H—

Haggard, Carl N., Infectious Hepatitis and Homologous Serum Jaundice 148
Hardy, H. H., Jr., Hypertrophic Pyloric Stenosis. (Barker, White, Hardy, Jr.) 307
Hargrove, Marion D., Myxedema. (Chanton, Hargrove) 546
Hargrove, U. S., The Management of Prostatism 521
Hart, William W., Sigmoid Sinus Thrombosis. (Farrior, Hart) 20
Hatchette, Stakely, Radiation Treatment in Cancer 316
Hauser, George H., Human Equine Encephalomyelitis, Eastern Type, in Louisiana 551
Hays, Jack R., Acute Mastoiditis—Secondary to Fracture of the Mastoid. (Vedrenne, Hays) 504
Heart Disease, A Review of Certain Aspects of the Pathologic Physiology of Heart Disease, by Roderick C. Webb 418
Heart Failure, Congestive, The Pathogenesis of the Edema of, by E. H. Bresler 297
Hematuria: Its Clinical Significance, by C. O. Frederick 517
(Hemometakinesia), The "Borrowing-Lending" Hemodynamic Phenomenon and Its Therapeutic Application in Peripheral Vascular Disturbances, by Thorpe Ray, George E. Burch, and Michael E. DeBakey 6

Henington, V. Medd, Drug Reactions and Their Treatment, with Special Reference to the Use of BAL in Heavy Metal Poisoning. (Kennedy, Henington) 209
Hepatitis, Infectious and Homologous Serum Jaundice, by Carl N. Haggard 148
Hoarseness—Its Possible Implications and Its Proper Management, by George J. Taquino 125
Holland, Ned W., Tracheotomy with Complication of Pneumothorax. (Thomas, Holland) 565
Hollis, Walter J., Peritoneoscopy. (Brown, Hollis) 498
Hopkins, Joseph V., Jr., Roentgenologic Aspect of Pneumonias. (Menville, Hopkins) 63
Hull, Edgar, Clinico-Pathologic Conference. (Dunlap, Hull) 324
Hull, Edgar, Medical Aspect of 6-Propylthiouracil. (Bain, Hull) 53
Human Equine Encephalomyelitis, Eastern Type, in Louisiana, by George H. Hauser 551
Human Infection with Virus of Equine Encephalomyelitis, Eastern Type, in Louisiana, by Kelsey C. Milner, G. Peyton Kelley and Morris F. Shaffer 270
Human Isoimmunization, by J. W. Davenport, Jr. 376
Hunt, Arthur B., Circumvallate Placenta. (Mussey, Faber, Hunt) 203
The Hutchinson Memorial Cancer Detection Clinic, by Robert L. Pye 72
Hypertrophic Pyloric Stenosis, by H. O. Barker, H. H. Hardy, Jr., and H. A. White 307
Hypothyroidism, Adult, and Myxedema, by Norman Burnstein 374

—I—

Impotence, by Paul L. Getzoff 77
Index to Volume 100, July, 1947—June, 1948 587
Infants, Intramedullary Infusions in, by Mack Sutton and Peyton Kelley 266
Infants, Postmortem Observations in Twenty-Two Premature, by James B. Arey 258
Infection, Enteric, or Infestation Sine Dysentery, by Daniel N. Silverman and Alan Leslie 101
Infectious Hepatitis and Homologous Serum Jaundice, by Carl N. Haggard 148
Infusions, Intramedullary, in Infants, by Mack Sutton and Peyton Kelley 266
In Memoriam 177
Intramedullary Infusions in Infants, by Mack Sutton and Peyton Kelley 266
Intravenous Ether (Diethyloxide), by O. C. Williams 470
Isaacson, Morris, Myocardial Aneurysm Causing Esophageal Obstruction 568
Isoimmunization, Human, by J. W. Davenport, Jr. 376

—J—

Jacobs, Sydney, Chronic Pneumonias 207
Jaundice, Congenital Hemolytic, by Robert Kapsinow 66
Jaundice, Homologous Serum and Infectious Hepatitis, by Carl N. Haggard 148
Johansen, Frederick, A., Sulfone Treatment of Leprosy. (Erickson, Wolcott, Johansen) 475
Joseph, Gerald F., Sulfonamides and Antibiotics in Otolaryngology 367

—K—

Kapsinow, Robert, Congenital Hemolytic Jaundice 66
Kelley, G. Peyton, Human Infection with Virus of Equine Encephalomyelitis, Eastern Type, in Louisiana. (Milner, Shaffer and Kelley) 270
Kelley, Peyton, Intramedullary Infusions in Infants. (Sutton, Kelley) 266
Kennedy, C. Barrett, Drug Reactions and Their Treatment, with Special Reference to the Use of BAL in Heavy Metal Poisoning. (Henington, Kennedy) 209
Kimbrough, Robert A., The Treatment of Uterine Prolapse 16
King, E. L., Saddle Block Anesthesia in Obstetrics. (Dyer, King) 70

—L—

Leprosy, The Chemotherapy of, by Herman Gray 218
Leprosy, Sulfone Treatment of, by Paul T. Erickson, Rolla R. Wolcott, and Frederick A. Johansen 475
Leslie, Alan, Enteric Infection or Infestation Sine Dysentery. (Silverman, Leslie) 101
Lippard, Vernon W., The Present Status of the Poliomyelitis Problem 262
Liver, Needle Biopsy of the, by W. D. Davis, Jr. 159
Loeber, Maud, Some Recent Experiences with Amebiasis in Children. (D'Antoni, Loeber) 276

Louisiana State Medical Society News.....45, 88, 136, 185, 245, 287, 339, 390, 440, 484, 532, 581
Loveman, David E., The Present Status of Our Knowledge Concerning the Spread of Poliomyelitis 449
Lyons, Champ, The Surgical Use of Chemotherapeutic and Antibiotic Agents..... 358

—Mc—

McHardy, Gordon, Papilloma of the Gallbladder. (Edwards, McHardy)..... 573
McLean, L. D., Pruritis Ani and Vulvae. (Van Studdiford, McLean)..... 445

—M—

Marrow, Sternal, in Health and Disease, by Phillip Pizzolato..... 3
Malaria, Present Day Treatment of, by A. J. Walker 95
The Management of Prostatism, by U. S. Hargrove 521
Mastoiditis, Acute—Secondary to Fracture of the Mastoid, by Clifford Vedrenne and Jack R. Hays..... 504
Medical Aspect of 6-Propylthiouracil, by Lamar Bain and Edgar Hull..... 53
Memoriam, In..... 177
Meningitis—Recent Advance in Therapy, by C. J. Tripoli and W. G. Unglaub..... 492
Menville, Leon J., Roentgenologic Aspect of Pneumonias. (Hopkins, Menville)..... 63
Metal Poisoning, Heavy, Drug Reactions and Their Treatment, with Special Reference to the Use of BAL in, by C. Barrett Kennedy andV. Medd Henington..... 209
Michel, Marshall L., The Diagnosis and Treatment of Acute Malignant Obstruction of the Large Bowel 397
Milner, Kelsey C., Human Infection with Virus of Equine Encephalomyelitis, Eastern Type, in Louisiana. (Kelley, Shaffer, Milner)..... 270
Mobilization, Rapid Postoperative, by Vincent D'Ingianni 80
Molnar, Joseph G., Epidemiology and Recent Developments in Poliomyelitis..... 408
Moore, Morris, Chromomycosis. (Mundt, Moore)..... 558
Mundt, Leslie K., Chromomycosis. (Moore, Mundt) 558
Mussey, Robert D., Circumvallate Placenta. (Hunt, Faber, Mussey)..... 203
Myocardial Aneurysm Causing Esophageal Obstruction, by Morris Isaacson..... 568
Myxedema and Adult Hypothyroidism, by Norman Burnstein..... 374
Myxedema, by Edwin F. Chanton and Marion D. Hargrove 546

—N—

Needle Biopsy of the Liver, by W. D. Davis, Jr..... 159
Nerve Paralysis, Facial, Resulting from Fracture of the Temporal Bone, by J. Brown Farrior and Pearson C. Caldwell..... 23
New Method of Treating Peptic Ulcers with Penicillin Gastric Drip, by Richard W. Young..... 379
Nose, Functional Conditions of the, by Joseph Stamm 322

—O—

Obstetrics, Saddle Block Anesthesia in, by Dr. E. L. King and Isadore Dyer..... 70
Ocular Anomalies, Congenital, Resulting From (a) Premature Birth (b) Rubella Infection of Mother during Pregnancy, by W. R. Buffington 466
Organization Section.....41, 85, 133, 179, 242, 283, 335, 388, 437, 481, 528, 579
Ovarian Sterility, A Survey of General and, with Special Reference to Anovulation, by Stonewall B. Stickney..... 26

—P—

Papilloma of the Gallbladder, by Gordon McHardy and Edwin Edwards..... 573
Paralysis, Facial Nerve, Resulting from Fracture of the Temporal Bone, by J. Brown Farrior and Pearson C. Caldwell..... 23
The Pathogenesis of the Edema of Congestive Heart Failure, by E. H. Bresler..... 297
Peptic Ulcer, The Etiology of, by Paul B. Daron..... 163
Peritoneoscopy, by Walter J. Hollis and Wallace H. Brown..... 498

Peters, Johan T., The Determination of Serum-and Urine-Salicyl Levels during Treatment with Salicyl-Drugs ... 1
Pizzolato, Philip, Sternal Marrow in Health and Disease ... 3
Placenta, Circumvallate, by Arthur B. Hunt, Robert D. Mussey, and John E. Faber ... 203
Pneumonias, Chronic, by Sydney Jacobs ... 207
Pneumonias, Roentgenologic Aspect of, by Joseph V. Hopkins, Jr. and Leon J. Menville ... 63
Pneumothorax, Tracheotomy with Complication of, by H. Ashton Thomas and Ned W. Holland ... 565
Poisoning, Lead, A Family Outbreak of, from Storage Battery Casings, by David Spizer ... 278
Poliomyelitis, by Branch J. Aymond ... 491
Poliomyelitis, Epidemiology and Recent Developments in, by Joseph G. Molnar ... 408
Poliomyelitis, The Present Status of Our Knowledge Concerning the Spread of, by David E. Loveman ... 449
Poliomyelitis Problem, The Present Status of the, by Vernon W. Lippard ... 262
Postmortem Observations in Twenty-Two Premature Infants, by James B. Arey ... 258
Postoperative Mobilization, Rapid, by Vincent D'Ingianni ... 80
Premature Infants, Postmortem Observations in Twenty-two, by James B. Arey ... 258
Prematurity—An Orientation, by Ethel C. Durham ... 119
Present Day Treatment of Malaria, by A. J. Walker ... 95
The Present Status of Our Knowledge Concerning the Spread of Poliomyelitis, by David E. Loveman ... 449
The Present Status of the Poliomyelitis Problem, by Vernon W. Lippard ... 262
Program for 1948 Annual Meeting ... 386
6-Propylthiouracil, Medical Aspect of, by Lamar Bain and Edgar Hull ... 53
Prostatism, The Management of, by U. S. Hargrove ... 521
Pruritis Ani and Vulvae, by M. T. Van Studdiford and L. D. McLean ... 445
Pye, Robert L., The Hutchinson Memorial Cancer Detection Clinic ... 72

—R—

Radiation Treatment in Cancer, by Stakely Hatchette ... 316
Radiotherapy for Gynecologic Cancer, by Manuel Garcia and Joseph V. Schlosser ... 141
Rapid Postoperative Mobilization, by Vincent D'Ingianni ... 80
Ray, Thorpe, The "Borrowing-Lending" Hemodynamic Phenomenon (Hemometakinesia) and Its Therapeutic Application in Peripheral Vascular Disturbances. (Burch, DeBakey, Ray) 6
Repair of Parotid Duct Fistula, by Leonard H. Stander and Claude C. Craighead ... 574
A Review of Certain Aspects of the Pathologic Physiology of Heart Disease, by Roderick C. Webb ... 418
The Rh Factor in Clinical Obstetrics and Gynecology, by J. W. Davenport, Jr. ... 510
Roentgenologic Aspect of Pneumonias, by Joseph V. Hopkins, Jr. and Leon J. Menville ... 63
Romano, Samuel A., The Use of Dicumarol in the Prevention and Treatment of Intravascular Clotting and Embolism ... 506

—S—

Saddle Block Anesthesia in Obstetrics, by E. L. King and Isadore Dyer ... 70
Salicyl-Drugs, The Determination of Serum-and Urine-Salicyl Levels during Treatment with, by Johan T. Peters ... 1
Salmon, George W., Airblock in the Newborn Period ... 253
Salmon, George W., Use of Antibiotics in Pediatrics ... 361
Schlosser, Joseph V., Radiotherapy for Gynecologic Cancer. (Garcia, Schlosser) ... 141
Shaffer, Morris F., Human Infection with Virus of Equine Encephalomyelitis, Eastern Type, in Louisiana. (Milner, Kelley, Shaffer) ... 270
Sharp, Robert F., Urological Complication Following Operation for Imperforate Anus ... 456
Sigmoid Sinus Thrombosis, by J. Brown Farrior and William W. Hart ... 20
Silverman, Arthur, The Wolff-Parkinson-White Syndrome (Bundle of Kent Syndrome; False Bundle Branch Block) ... 428
Silverman, Daniel N., Enteric Infection or Infestation Sine Dysentery. (Leslie, Silverman) ... 101
Sinus Thrombosis, Sigmoid, by J. Brown Farrior and William W. Hart ... 20
Some Recent Experiences with Amebiasis in Children, by Maud Loeber and Joseph S. D'Antoni 276
Spine Fusion, Lumbosacral, Double Notched Graft for, by Alfons R. Altenberg ... 464
Spizer, David, A Family Outbreak of Lead Poisoning from Storage Battery Casings ... 278

Stander, Leonard H., Repair of Parotid Duct Fistula. (Craighead, Stander) 574
Stamm, Joseph, Functional Conditions of the Nose 322
Stenosis, Hypertrophic Pyloric, by H. O. Barker, Jr., H. H. Hardy and H. W. White 307
Sterility, A Survey of General and Ovarian with Special Reference to Anovulation, by Stonewall B. Stickney 26
Sternal Marrow in Health and Disease, by Philip Pizzolato 3
Stickney, Stonewall B., A Survey of General and Ovarian Sterility with Special Reference to Anovulation 26
Stone, Harry A., The Use of Curare in Tetanus. (Stone, Browne) 382
Sulfonamides and Antibiotics in Otolaryngology, by Gerald F. Joseph 367
Sulfone Treatment of Leprosy, by Paul T. Erickson, Rolla R. Wolcott, and Frederick A. Johansen 475
The Surgical Use of Chemotherapeutic and Antibiotic Agents, by Champ Lyons 358
A Survey of General and Ovarian Sterility with Special Reference to Anovulation, by Stonewall B. Stickney 26
Sutton, Mack, Intramedullary Infusions in Infants. (Kelley, Sutton) 266
Symposium on Chemotherapy and Antibiotics—Chemotherapy and Antibiotics, by Maxwell Finland 345

—T—

Taquino, George J., Hoarseness—Its Possible Implications and Its Proper Management 125
Temporal Bone, Facial Nerve Paralysis Resulting from Fracture of the, by J. Brown Farrior and Pearson C. Caldwell 23
Tetanus, The Use of Curare in, by E. Zollicoffer Browne and Harry A. Stone 383
Thiouracil in Toxic Thyroid Disease, by Frederick Fitzherbert Boyce and Donald D. Dieter 56
Thomas, H. Ashton, Tracheotomy with Complication of Pneumothorax. (Holland, Thomas) 565
Thrombosis, Sigmoid Sinus, by J. Brown Farrior and William W. Hart 20
Thyroid Disease, Thiouracil in Toxic, by Frederick Fitzherbert Boyce and Donald D. Dieter 56
Tracheotomy with Complication of Pneumothorax, by H. Ashton Thomas and Ned W. Holland 565
Transactions of Orleans Parish Medical Society 44, 87, 135, 183, 286, 337
The Treatment of Chronic Anemia, by Norton W. Voorhies 310
The Treatment of Uterine Prolapse, by Robert A. Kimbrough 16
Tripoli, C. J., Meningitis—Recent Advances in Therapy. (Unglaub, Tripoli) 492

—U—

Ulcer, Peptic, The Etiology of, by Paul B. Daron 163
Ulcers, Peptic, New Method of Treating with Penicillin Gastric Drip, by Richard W. Young 379
Unglaub, W. G., Meningitis—Recent Advances in Therapy. (Tripoli, Unglaub) 492
Urological Complication Following Operation for Imperforate Anus, by Robert F. Sharp 456
Use of Antibiotics in Pediatrics, by George W. Salmon 361
The Use of Curare in Tetanus, by E. Zollicoffer Browne and Harry A. Stone 383
The Use of Dicumarol in the Prevention and Treatment of Intravascular Clotting and Embolism, by Samuel A. Romano 506
Uterine Prolapse, The Treatment of, by Robert A. Kimbrough 16

—V—

Van Studdiford, M. T., Pruritis Ani and Vulvae (McLean, Van Studdiford) 445
Vedrenne, Clifford, Acute Mastoiditis—Secondary to Fracture of the Mastoid. (Hays. Vedrenne) 504
Voorhies, Norton W., The Treatment of Chronic Anemia 310

—W—

Walker, A. J., Present Day Treatment of Malaria 95
Webb, Roderick C., A Review of Certain Aspects of the Pathologic Physiology of Heart Disease 418
White, H. A., Hypertrophic Pyloric Stenosis. (Barker, Hardy, Jr., White) 307
Williams, O. C., Intravenous Ether (Diethyloxide) 470
Wolcott, Rolla R., Sulfone Treatment of Leprosy. (Erickson, Johansen, Wolcott) 475

The Wolff-Parkinson-White Syndrome (Bundle of Kent Syndrome; False Bundle Branch Block), by Arthur Silverman 428
Woman's Auxiliary to the Louisiana State Medical Society 192
Woody, Norman C., Farber's Test in the Diagnosis of Congenital Intestinal Atresia 273

—Y—

Young, Richard W., New Method of Treating Peptic Ulcers with Penicillin Gastric Drip 379
Young, Roy Carl, Clinical Observations on the Treatment of the Alcoholic 539